
Foodborne Illness, Australia, Circa 2000 and Circa 2010

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Foodborne disease is a major public health problem worldwide. To examine changes in foodborne illness in Australia, we estimated the incidence, hospitalizations, and deaths attributed to contaminated food circa 2010 and recalculated estimates from circa 2000. Approximately 25% of gastroenteritis cases were caused by contaminated food; to account for uncertainty we used simulation techniques to estimate 90% credible intervals. We estimate that circa 2010, 4.1 million foodborne gastroenteritis cases occurred, and circa 2000, 4.3 million cases occurred. Circa 2010, contaminated food was estimated to be responsible for 30,840 gastroenteritis-associated hospitalizations, 76 associated deaths, and 5,140 nongastrointestinal illnesses. Cases of salmonellosis and campylobacteriosis increased from 2000 to 2010 and were the leading causes of gastroenteritis-associated hospitalizations; *Listeria monocytogenes* and nontyphoidal *Salmonella* spp. infections were the leading causes of death. Although the overall incidence of foodborne illnesses declined over time in Australia, cases of foodborne gastroenteritis are still common.

Foodborne illness is a major public health problem and a common cause of illness and death worldwide. Outbreaks linked to contaminated food can affect the public's trust and financially harm implicated businesses and associated food industries. Estimates of the effects of foodborne illnesses and individual pathogens provide evidence for policy interventions and food safety regulation. In addition, estimates of changes in the incidence of foodborne illnesses and hospitalizations over time provide information on the effectiveness of changes to food safety standards and regulation.

Many agents can cause foodborne illness; some of these agents are transmitted to humans by other routes as well as by food. Most foodborne illnesses manifest as gastroenteritis, but other presentations, such as meningitis and hepatitis may also result from infection, and sequelae may occur weeks after the acute infection.

Many countries have estimated the incidence of foodborne diseases (1–5). In Australia in 2000, foodborne

incidence, hospitalizations, and deaths were estimated to cost 1.25 billion Australian dollars annually (6,7). However, since 2000, surveillance has substantially improved, data availability has increased, and methods have been refined. To inform current public health decisions and policies in Australia, we used new methods and datasets to estimate the incidence of infectious gastroenteritis and associated hospitalizations and deaths in Australia circa 2010. We then applied these refined methods to circa 2000 data so that estimates from the 2 periods could be directly compared.

Methods

We estimated the incidence of illness and the number of hospitalizations and deaths associated with 23 potentially foodborne pathogens or agents in Australia circa 2010 (online Technical Appendix 1 Table 1, <http://wwwnc.cdc.gov/EID/article/2011/13-1315-Techapp1.pdf>). Pathogens we did not consider relevant were those acquired only overseas (e.g., *Vibrio cholerae*, *Trichinella spiralis*) and those that cause gastroenteritis but are not proven agents of foodborne disease (e.g., *Clostridium difficile*). Estimates of chronic sequelae from foodborne illnesses are discussed elsewhere in this issue (8).

When possible, data for the circa 2010 study period covered 2006–2010, and all denominator data were based on the Australian population during that period (9). Estimates of incidence relied on data obtained from 4 sources: notifiable disease surveillance at the national and state levels; outbreak surveillance through the OzFoodNet Outbreak Register; the National Gastroenteritis Survey II (NG-SII; <http://www.ozfoodnet.gov.au/>), a cross-sectional survey; and the Water Quality Study (WQS), a randomized controlled trial (conducted during 1997–1999) of household water treatment to prevent gastroenteritis (10,11). Estimates of severe illness were determined by using hospitalization and death data. This study was approved by the Australian National University Human Research Ethics Committee. Further details of the data sources and methods are in online Technical Appendix 1.

To estimate incidence, hospitalizations, and deaths, we built on our previous methods (7), making them similar to those used in the United States (2,3). We calculated estimates

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by using simulation techniques in @Risk version 6 (<http://www.palisade.com/>) with multiple inputs, each with different levels of uncertainty. We used empirical, lognormal, and PERT (program evaluation review technique) probability distributions to model uncertainty in source data and multipliers. Estimates are expressed as probability distributions summarized by a median point estimate with a 90% credible interval (CrI) (online Technical Appendix 2, <http://wwwnc.cdc.gov/EID/article/2011/13-1315-Techapp2.pdf>).

Incidence Circa 2010

To estimate the annual incidence of infectious gastroenteritis in Australia circa 2010, we used symptoms included in the NGSII telephone survey conducted during February 2008–January 2009. Case definition has a considerable effect when determining the incidence of gastroenteritis (12). To enable a valid comparison of circa 2000 and circa 2010 gastroenteritis estimates, we used the case definition from the earlier study (13,14). In NGSII, persons were considered case-patients if they had ≥ 3 episodes of diarrhea or ≥ 2 episodes of vomiting within a 24-h period during the preceding 4 weeks and did not report a noninfectious cause for their illness. However, for persons who had concomitant respiratory symptoms, we applied a stricter definition: ≥ 4 episodes of diarrhea and/or ≥ 3 episodes of vomiting (15). In NGSII, 4.5% (341/7,578) of survey respondents reported gastroenteritis in the preceding 4 weeks, equating to 0.74 gastroenteritis episodes per person per year (95% CI 0.64–0.84) or 15.9 million cases annually in Australia.

We used 2 main approaches to estimate the incidence of foodborne illness caused by specific pathogens or illnesses. Our preferred approach was the surveillance approach, in which we estimated the community incidence of illness by applying an underreporting multiplier to scale up data from notifiable disease surveillance. When these data were not available, we used a pathogen fraction approach, in which we estimated the percentage of overall gastroenteritis caused by specific pathogens. When data were unavailable by either of these approaches, we used other surveillance data, such as outbreak data. Approach-specific flow charts are provided in online Technical Appendix 2.

Using the surveillance approach, we adjusted for underreporting of community cases to public health surveillance. We used findings from an underreporting multiplier study in Australia (16) for moderate illnesses and bloody diarrhea. For serious illnesses, we assumed the underreporting factor as 1 illness reported for every 2 that occurred in the community, as used by Mead et al. (17) and Scallan et al. (2). We applied another multiplier to outbreak surveillance data to adjust for underreporting when only outbreak cases were notified (online Technical Appendix 2).

When we used the pathogen fraction approach, our main data source was the WQS (10,11). The WQS provided

data on the proportion of gastroenteritis episodes caused by specific pathogens, and we applied those proportions to total foodborne illness incidence data from the NGSII. However, the WQS was conducted before rotavirus vaccine was added to the Australian vaccination schedule. To account for the effect of the vaccine on infection incidence, we calculated a time-trend multiplier by using age-specific hospitalization data from before and after introduction of rotavirus vaccine (18).

We used the surveillance approach for cases caused by 16 pathogens, of which 11 were from the National Notifiable Diseases Surveillance System (NNDSS; <http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-nndssintro.htm>) and 5 were from outbreak data. We used the pathogen fraction approach for cases caused by 6 pathogens. In addition, because local data were lacking, we applied US seroprevalence data to the Australian population data to estimate the incidence of toxoplasmosis (online Technical Appendix 2).

Incidence Circa 2000

Methods for calculating incidence have changed since the circa 2000 estimates were determined (7); the changes include updated underreporting multipliers (16), more rigorous expert elicitation (19), and new estimates of the foodborne multipliers for some pathogens. These changes could result in a potentially misleading comparison of circa 2010 and circa 2000 findings. We recalculated estimates for circa 2000 by using the original data with methods identical to those used for circa 2010 data. Updated estimates of the total incidence of foodborne gastroenteritis were determined by using the original 2001 National Gastroenteritis Survey, together with the 2010 foodborne proportion of 25% (compared with 32% in the circa 2000 study). To recalculate the circa 2000 estimates, we replaced multipliers used in that study with circa 2010 multipliers and applied them to 1996–2000 data from NNDSS for *Campylobacter* spp., nontyphoidal *Salmonella enterica* serotypes (hereafter referred to as nontyphoidal *Salmonella* spp.), *S. enterica* serotype Typhi, *Shigella* spp., hepatitis A virus, and *Listeria monocytogenes* infections and to 1996–2000 surveillance data from the state of Victoria, Australia, for *Giardia lamblia*. Only pathogens for which we had surveillance data from both periods were included in this analysis.

Hospitalizations and Deaths

We estimated the annual number of hospitalizations for foodborne illnesses by using 2006–2010 state and territory hospitalization data (<http://www.aihw.gov.au/hospitals/australian-hospital-statistics/>) for which principal and additional diagnoses were based on the Australian modification of the 10th International Classification of Diseases (20), and we estimated the annual number of deaths by using 2001–2010

Australian Bureau of Statistics' national death data for underlying or contributing cause (<http://www.aihw.gov.au/deaths/>). Reports for a large number of hospitalizations and deaths caused by gastrointestinal illnesses that were presumed infectious did not identify a specific pathogen.

We adjusted for travel-associated cases and estimated the proportions of foodborne disease-associated hospitalizations and deaths (online Technical Appendix 3, <http://wwwnc.cdc.gov/EID/article/2011/13-1315-Techapp3.pdf>). Because the recorded hospitalizations and deaths associated with each pathogen reflect only laboratory-confirmed cases, we applied an underdiagnosis multiplier of 2 (range 1–3). This multiplier has been used in other studies (2,7,17) but never validated. Assuming that outbreaks provide a representative denominator population from which to calculate the proportion of hospitalized case-patients, we confirmed the appropriateness of the multiplier by using the OzFoodNet Outbreak Register (<http://www.ozfoodnet.gov.au/>) to calculate, for a number of pathogens, the proportion of hospitalized case-patients. For the included pathogens, we compared this proportion with the ratio of our estimated yearly hospitalizations to yearly illnesses.

Domestically Acquired Multiplier

To exclude infections acquired overseas, we applied a domestically acquired multiplier to all pathogens to adjust the total incidence data. For many pathogens, this multiplier was estimated from surveillance data from states and territories that recorded illnesses acquired overseas; variability by state and by year was used to inform uncertainty in the multiplier. Other pathogens causing illness of short duration were assumed to be 100% domestically acquired. Details, by pathogen, are provided in online Technical Appendix 4 (<http://wwwnc.cdc.gov/EID/article/2011/13-1315-Techapp4.pdf>).

Proportion Foodborne Multiplier

To estimate the total number of foodborne infections caused by each pathogen, we applied a pathogen-specific proportion foodborne multiplier to all pathogens (online Technical Appendix 2 Table 2). The proportion foodborne multiplier was estimated for 9 pathogens in 2009 by using an expert elicitation process (19), and the multipliers for another 9 pathogens were estimated by using a similar expert elicitation study in 2005 (21). All illnesses due to seafood toxins were assumed to be caused by food, and multipliers for 3 viruses were assumed to be equal to those for similar pathogens.

The estimated annual number of gastroenteritis cases caused by 18 known pathogens/parasites for the circa 2010 study period is listed in Table 1. An estimated 25% of the cases were caused by contaminated food, of which 36%, 16%, and 11% were caused by bacteria, viruses, and parasites,

respectively. Given an absence of other data sources, we applied this overall foodborne proportion of 25% to the total number of gastroenteritis cases to determine the number caused by contaminated food (3,17,22).

Results

Incidence

Foodborne Gastroenteritis Circa 2010

We estimated that each year circa 2010, 4.1 million domestically acquired cases (90% CrI 2.3–6.4) of foodborne gastroenteritis occurred in Australia. Of those annual cases, 0.8 million were caused by the 18 pathogens that were known agents of gastroenteritis, and the remaining 3.3 million cases were caused by unknown or unidentified pathogens (Table 1; online Technical Appendix 5, <http://wwwnc.cdc.gov/EID/article/2011/13-1315-Techapp5.pdf>). Pathogenic *Escherichia coli*, norovirus, *Campylobacter* spp., and nontyphoidal *Salmonella* spp. were the most common causes of foodborne gastroenteritis; together, they were responsible for 93% of the foodborne illnesses caused by known pathogens.

Foodborne Nongastrointestinal Illness Circa 2010

In addition to causing foodborne gastroenteritis, contaminated food also caused 5,140 cases (90% CrI 3,530–7,980) of nongastrointestinal illness in Australia circa 2010 (Table 2). Toxoplasmosis was the most common foodborne nongastrointestinal illness; 3,750 cases (90% CrI 1,400–7,150) occurred each year. The percentage of foodborne illnesses caused by nongastroenteric agents ranged from a low of 12% for hepatitis A infection to a high of 100% for scombrototoxicosis and ciguatera.

Comparison of Circa 2010 Estimates with Circa 2000 Estimates

When we applied the newer estimation methods, including the new proportion foodborne multiplier (i.e., 25%), to circa 2000 data, the annual number of foodborne gastroenteritis cases was 4.3 million (90% CrI: 2.2–7.3). That total translates to a circa 2000 incidence of 224,000 cases/million population (90% CrI 116,000–374,000). Comparison of the circa 2010 incidence (186,000 cases/million population; 90% CrI 105,000–289,000) with the circa 2000 incidence showed a 17% decreased incidence of foodborne gastroenteritis between 2000 and 2010, although the CrI included 1 (rate ratio [RR] 0.83, 90% CrI 0.4–1.8). Similar recalculation of circa 2000 estimates for key gastrointestinal pathogens showed a total of 28,000 cases (90% CrI 15,000–50,000) of foodborne salmonellosis each year (incidence 1,500 cases/million population, 90% CrI 800–2,700) and 139,000 cases (90% CrI 82,500–227,000) of

Table 1. Estimated number of gastroenteritis cases caused by domestically acquired pathogens, Australia, circa 2010*

Causative agent	Total no. cases, median (90% CrI)	% Cases caused by contaminated food, median (90% CrI)	No. cases caused by contaminated food, median (90% CrI)
Bacterium			
<i>Bacillus cereus</i>	3,350 (900–10,100)	100 (98–100)	3,350 (900–10,100)
<i>Campylobacter</i> spp.	234,000 (147,000–374,000)	77 (62–89)	179,000 (108,500–290,000)
<i>Clostridium perfringens</i>	16,500 (2,600–53,400)	98 (86–100)	16,100 (2,550–50,600)
STEC	4,300 (2,050–9,500)	56 (32–83)	2,350 (950–5,850)
Other pathogenic <i>E. coli</i>	1,100,000 (833,000–1,450,000)	23 (8–55)	255,000 (85,800–632,000)
<i>Salmonella</i> spp, nontyphoidal	56,200 (31,900–101,000)	72 (53–86)	39,600 (21,200–73,400)
<i>Salmonella enterica</i> ser. Typhi	20 (8–45)	75 (2–97)	15 (5–30)
<i>Shigella</i> spp.	3,000 (1,650–5,400)	12 (5–23)	350 (150–850)
<i>Staphylococcus aureus</i>	1,300 (200–7,050)	100 (95–100)	1,300 (200–7,000)
<i>Vibrio parahaemolyticus</i>	60 (15–170)	75 (5–96)	40 (10–120)
<i>Yersinia enterocolitica</i>	1,500 (900–2,500)	84 (28–94)	1,150 (650–1,950)
Virus			
Adenovirus	88,400 (28,800–205,000)	2 (1–3)	1,650 (500–4,650)
Astrovirus	67,100 (20,900–155,000)	2 (1–3)	1,300 (350–3,400)
Norovirus	1,550,000 (1,220,000–1,940,000)	18 (5–35)	276,000 (78,100–563,000)
Rotavirus	44,800 (18,500–90,800)	2 (1–3)	850 (300–2,000)
Sapovirus	81,600 (63,400–102,000)	18 (5–35)	15,000 (7,450–24,300)
Parasite			
<i>Cryptosporidium</i> spp.	17,900 (8,150–39,800)	10 (1–27)	1,700 (150–6,100)
<i>Giardia lamblia</i>	32,800 (19,800–56,400)	6 (1–50)	3,700 (800–10,600)
Subtotal	3,090,000 (2,810,000–3,900,000)	25 (13–42)	798,000 (528,000–1,310,000)
Unknown etiology	12,800,000 (10,500,000–14,500,000)	25 (13–42)	3,310,000 (1,800,000–5,152,000)
Total	15,900,000 (13,700,000–18,000,000)	25 (13–42)	4,110,000 (2,330,000–6,390,000)

*All estimates were based on an empirical distribution of the Australian population in the June quarter of 2006–2010; for the parameters of these distributions, see online Technical Appendix 4 (<http://wwwnc.cdc.gov/EID/article/2011/13-1315-Techapp4.pdf>). CrI, credible interval; *E. coli*, *Escherichia coli*; STEC, Shiga toxin-producing *E. coli*.

foodborne campylobacteriosis each year (incidence 7,400 cases/million population, 90% CrI 4,500–12,200) (Table 3). Comparison of the circa 2000 and circa 2010 incidence rates showed RRs of 1.24 (90% CrI 0.5–2.8) for foodborne salmonellosis and 1.13 (90% CrI 0.5–2.3) for foodborne campylobacteriosis, although the CrI included 1. CrIs include uncertainty derived from incidence multipliers and were considerably wider than intervals for ratios derived from raw surveillance data.

Hospitalizations

Circa 2010, there were an estimated 30,600 hospitalizations (90% CrI 28,000–34,000) for foodborne gastroenteritis and 240 hospitalizations (90% CrI 180–350) for nongastrointestinal foodborne illnesses (Table 4). Approximately 5,900 of all hospitalizations for gastroenteritis were for illnesses caused by known pathogens, of which *Campylobacter* spp. and nontyphoidal *Salmonella* spp. were the leading causes of hospitalization, and *L. monocytogenes* was the leading cause of nongastrointestinal illnesses requiring hospitalization. The remaining 24,700 hospitalizations were for gastroenteritis of unknown etiology.

Deaths

For circa 2010, we estimated that there were 60 deaths (90% CrI 53–63) due to foodborne gastroenteritis and 16 deaths (90% CrI 10–21) due to nongastrointestinal foodborne illnesses (Table 4). Nontyphoidal *Salmonella* spp.

and *L. monocytogenes* were the most commonly identified causes of all illnesses that resulted in death; each year, these pathogens were each responsible for an estimated 15 foodborne illness-associated deaths. Gastroenteritis of unknown etiology as an underlying or contributing cause of death resulted in 39 deaths each year.

Discussion

Foodborne illness is extremely common in Australia: on average, each person in Australia experiences an episode of foodborne gastroenteritis approximately every 5 years. Although foodborne gastroenteritis is often not serious, the cost to society is considerable through direct medical costs and days of lost work. Approximately 1 in 5 persons with gastroenteritis seeks medical attention. Thus, up to 1 million medical visits a year could be for foodborne illnesses (23).

We examined changes in foodborne illness in Australia over time, a key reason for repeating studies to estimate incidence. Our findings showed a slight decline in the rate of foodborne gastroenteritis between the circa 2000 and circa 2010 study periods, but our findings also showed increases in the rates of illness caused by some specific pathogens. Changed estimates were driven by differences in estimates of total gastroenteritis and by pathogen-specific surveillance trends. In Australia from 2006 onward, the number of raw egg-associated salmonellosis outbreaks has markedly increased (24), and since 2000, the numbers of

Table 2. Estimated number of acute foodborne illness cases caused by domestically acquired pathogens and agents that do not result in gastroenteritis, Australia, circa 2010*

Illness	% Foodborne, median (90% CrI)	No. illnesses, median (90% CrI)
Hepatitis A virus infection	12 (5–24)	40 (10–100)
Listeriosis	98 (90–100)	150 (50–200)
Toxoplasmosis	31 (4–74)	3,750 (1,400–7,150)
Ciguatera	100 (100–100)	150 (40–300)
Scombrototoxicosis	100 (100–100)	1,050 (0–2,450)
Total	40 (25–59)	5,140 (3,530–7,980)

*All estimates were based on an empirical distribution of the Australian population in the June quarter of 2006–2010; for the parameters of these distributions, see online Technical Appendix 4 (<http://wwwnc.cdc.gov/EID/article/20/11/13-1315-Techapp4.pdf>). CrI, credible interval.

notified laboratory-confirmed cases of campylobacteriosis and salmonellosis have increased (25). Estimates of rotavirus cases for circa 2010 were lower than those for circa 2000, reflecting the success of the vaccination program (18). Also, the estimated number of foodborne illness cases caused by hepatitis A virus declined from 245 cases/year circa 2000 to 40 cases/year circa 2010, reflecting improved disease control through vaccination (24). Although these interventions were not targeted at foodborne disease, our findings highlight the benefits of vaccination programs in reducing circulation of enteric pathogens and transmission through food.

It must be noted that where we observed changes over time, they were often not significant due to the many sources of uncertainty. When we examined the CrIs, over half of the uncertainty arose from the distribution for the foodborne multiplier estimated from expert elicitation; most of the other sources of uncertainty arose from the distributions for the underreporting and pathogen fraction multipliers. Further studies to estimate foodborne multipliers for high-incidence pathogens (in particular, norovirus and other pathogenic *E. coli*) would help reduce this uncertainty in overall estimates. Scallan et al. (3) highlighted the profound effect that changes in these proportions of foodborne transmission can have on overall estimates of disease incidence. We identified similar effects when we used updated methods to recalculate estimates for circa

2000; in particular, the estimates for foodborne gastroenteritis illnesses declined from 5.4 to 4.3 million cases. New approaches should be examined for estimating the relative importance of different modes of transmission for pathogens that are potentially foodborne.

Similar studies estimating the incidence of foodborne disease have been conducted in the United States (2,3,17), United Kingdom (4), Canada (22), and the Netherlands (5). We estimated that 25% of all gastroenteritis cases in Australia were caused by contaminated food; this percentage is similar to estimates for the United Kingdom and to the most recent estimates for the United States but lower than estimates for the Netherlands. Although the Canadian study does not report an overall proportion of foodborne transmission, analysis of the study results puts it at ≈20% (22). In the United States, Scallan et al. (2) estimated that 9.4 million (26%) of 36.4 million domestically acquired illnesses caused by known pathogens were transmitted via contaminated food, and in the United Kingdom, Adak et al. (4) estimated that 26% of infectious intestinal illnesses were caused by pathogens transmitted via contaminated food. The estimate for the Netherlands was higher at 39% (5). These overall estimates of the proportion of gastroenteritis caused by contaminated food depend on the pathogens included in the estimates, the incidence of common pathogens in the study area, and the proportion of those common pathogens that are considered to be foodborne.

Table 3. Comparison of estimates of the annual number of cases and incidence rates for foodborne gastroenteritis and key foodborne pathogens, Australia, circa 2000 and circa 2010*

Foodborne illness/pathogen	Circa 2000		Circa 2010		RR (90% CrI)
	No. cases, median (90% CrI)	Rate per million population (90% CrI)	No. cases, median (90% CrI)	Rate per million population (90% CrI)	
Gastroenteritis	4.3 million (2.2–7.3 million)	224,000 (116,000–374,000)	4.1 million (2.3–6.4 million)	186,000 (105,000–289,000)	0.83 (0.4–1.8)
<i>Campylobacter</i> spp.	139,000 (82,500–227,000)	7,400 (4,500–12,200)	179,000 (108,500–290,000)	8,400 (5,050–13,650)	1.13 (0.5–2.3)
<i>Salmonella</i> spp., nontyphoidal	28,000 (15,000–50,000)	1,500 (800–2,700)	39,600 (21,200–73,400)	1,850 (1,000–3,350)	1.24 (0.5–2.8)
<i>Salmonella enterica</i> ser. Typhi	9 (3–21)	0.5 (0–1)	15 (5–30)	0.6 (0–1)	1.2 (0.5–2.6)
<i>Shigella</i> spp.	515 (175–1,300)	28 (9–70)	350 (150–850)	16 (6–40)	0.57 (0.2–2.3)
Hepatitis A virus	245 (65–725)	13 (3–40)	40 (10–100)	2 (1–5)	0.15 (0.06–0.4)
<i>Listeria monocytogenes</i>	125 (70–185)	7 (4–10)	150 (50–100)	7 (3–10)	1 (0.4–1.9)
<i>Giardia lamblia</i>	2,600 (565–7,400)	140 (30–405)	3,700 (800–10,600)	175 (35–490)	1.25 (0.5–1.9)

*Estimates are based on an empirical distribution of the Australian population in the June quarter of 1996–2000 (circa 2000 estimates) and 2006–2010 (circa 2010 estimates); for the parameters of these distributions, see online Technical Appendix 4 (<http://wwwnc.cdc.gov/EID/article/20/11/13-1315-Techapp4.pdf>). CrI, credible interval; RR, rate ratio.

Table 4. Estimated annual number of hospitalizations and deaths resulting from domestically acquired foodborne pathogens, parasites, and diseases, Australia, circa 2010*

Illness, causative agent/illness	ICD-10-AM code	No. hospitalizations, median (90% CrI)	No. deaths, median (90% CrI)
Gastrointestinal illness, cause			
Bacterium			
<i>Bacillus cereus</i>	A05.4	25 (4–45)	0
<i>Campylobacter</i> spp.	A04.5	3,200 (2,100–4,500)	3 (2–4)
<i>Clostridium perfringens</i>	A05.2	0 (0–2)	1 (0–1)
STEC	A04.3	7 (2–15)	0
Other pathogenic <i>E. coli</i>	A04.0, A04.1, A04.4	20 (6–50)	0 (0–1)
<i>Salmonella</i> spp., nontyphoidal	A02.0–A02.9	2,100 (1,300–3,000)	15 (8–20)
<i>Salmonella enterica</i> ser. Typhi	A01.0	15 (6–35)	0
<i>Shigella</i> spp.	A03	25 (9–50)	0
<i>Staphylococcus aureus</i>	A05.0	10 (7–20)	0
<i>Vibrio parahaemolyticus</i>	A05.3	1 (0–1)	0
<i>Yersinia enterocolitica</i>	A04.6	35 (10–65)	1 (0–1)
Virus			
Adenovirus	A08.2	15 (8–25)	0
Astrovirus	NA	NA	NA
Norovirus	A08.1	150 (35–350)	1 (0–2)
Rotavirus	A08.0	50 (30–100)	0 (0–0)
Sapovirus	NA	NA	NA
Parasite			
<i>Cryptosporidium</i> spp.	A07.2	40 (6–100)	0
<i>Giardia lamblia</i>	A07.1	100 (25–300)	0
Subtotal		5,900 (4,700–7,500)	21 (14–26)
Unknown etiology	A08.4, A09, A09.0, A09.9	24,700 (22,600–27,800)	39 (27–54)
Total		30,600 (28,000–34,000)	60 (53–63)
Nongastrointestinal illness			
Hepatitis A	B15.9	20 (6–50)	0 (0–2)
Listeriosis	A32	150 (100–250)	15 (9–20)
Toxoplasmosis	B58	30 (10–60)	1 (0–2)
Ciguatera	T61.0	25 (10–40)	0
Scombrototoxicosis	T61.1	8 (5–10)	0
Total		240 (180–350)	16 (10–21)

*All estimates based on an empirical distribution of the Australian population in the June quarter of 2006–2010 for hospitalizations and 2001–2010 for death; see online Technical Appendix 3 (<http://wwwnc.cdc.gov/EID/article/2011/13-1315-Techapp3.pdf>) for the methods used to determine these estimates. CrI, credible interval; ICD-10-AM, Australian modification of the 10th International Classification of Diseases; NA, not applicable. *E. coli*, *Escherichia coli*; STEC, Shiga toxin-producing *E. coli*.

The methods we used to calculate estimates in this study were refined from those used for the circa 2000 study, and in the intervening years, surveillance has improved and data availability has increased. In addition, we used national data to incorporate variations in foodborne disease patterns to provide more representative estimates. A further improvement was our use of more detailed hospitalization data. Previous hospitalization estimates for foodborne gastroenteritis were determined by using the hospital principal diagnosis data with a multiplier to adjust for additional diagnoses. In this study, we used the principal plus additional diagnoses data so that we could identify different diagnosis patterns by pathogen; for example, we found that 77% of the hospital diagnoses for salmonellosis were listed as principal diagnoses, whereas 37% of the diagnoses for norovirus infection were listed as principal diagnoses. Our new approach better captures different diagnosis patterns, especially for illnesses with multiple concomitant conditions (e.g., listeriosis) (26).

We also incorporated new expert elicitations into our methods to determine the circa 2010 estimates, further

improving data quality (19). These expert elicitations were undertaken in 2009 to decide which pathogens/agents should be included in the estimates and to determine the proportion of cases caused by foodborne transmission. Compared with estimates obtained by using the Delphi process in 2005 (21), the estimated proportion of foodborne transmission in the circa 2010 study was generally lower, and uncertainty bounds were generally wider. In particular, our estimates showed a lower proportion of foodborne transmission for *Clostridium perfringens*, other pathogenic *E. coli*, norovirus, nontyphoidal *Salmonella* spp., and Shiga toxin-producing *E. coli* (STEC). This finding may reflect that environmental sources of gastrointestinal infection have been somewhat neglected and that health departments have a primary focus on foodborne diseases (19). Compared with previously published estimates for 2000 (7), our estimates for circa 2000 showed fewer illnesses attributed to food; this difference was due to our use of lower foodborne proportions for some pathogens.

When estimating the community incidence of foodborne illness, we used underreporting multipliers to adjust

for the proportion of infected persons who did not seek treatment or submit specimens for testing. We used previously published estimates (16) of pathogen-specific multipliers for nontyphoidal *Salmonella* spp., *Campylobacter* spp., and STEC. The underreporting multiplier used for nontyphoidal *Salmonella* spp. (7, 95% CrI 4–14) was extrapolated to all other moderate illnesses, except *Campylobacter* spp. and STEC. These new underreporting multipliers were smaller than those used in previously published estimates for Australia (15, 95% CrI 5–25) (7).

The underreporting multiplier for serious illnesses and the underdiagnosis multiplier for hospitalizations and deaths remained at 2 (CrI 1–3), consistent with usage in other studies (2,17,27). The use of this multiplier for hospitalizations and deaths was validated by comparing data from the OzFoodNet Outbreak Register with hospital and death data, which suggested that a multiplier of at least 2 was necessary to account for underdiagnosis. Data on pathogen-specific underdiagnosis are limited, and further studies are required to thoroughly validate this multiplier and assess whether there are pathogen-specific differences in the underdiagnosis of severe illness.

The incidence of cases, hospitalizations, and deaths associated with foodborne pathogens in Australia does not show the complete burden from these pathogens because infection with some of them (i.e., *Campylobacter* spp., nontyphoidal *Salmonella* spp., and STEC) may lead to sequelae. The estimates in this study, together with our estimates of sequelae (8), highlight the considerable effect of foodborne *Campylobacter* spp. infection in Australia (28).

In a complex study of this type, there are several gaps and limitations in the data. While NNDSS and the OzFoodNet Outbreak Register are nationally representative, jurisdictions may have reported or coded their data differently. In addition, there were no available Australian data on toxoplasmosis, so we relied on data from the United States (29). We used data from the WQS (10,11) for pathogens that were not nationally notifiable or had limited outbreak data. The WQS study was the best of its kind in Australia; however, the data are now >15 years old, and the study population was based on families in Melbourne with children. We adjusted WQS data for changes over time and weighted the data for the age structure of the general population (online Technical Appendix 2). In addition, cohort study participants may be reluctant to provide fecal samples; in the WQS, only one third of persons with gastroenteritis submitted a fecal sample (11). Furthermore, the WQS did not test for all known foodborne pathogens, and a pathogen was identified for only 17% of the fecal specimens that were examined (10).

The estimated incidence of foodborne disease in Australia circa 2010 was considerable: 4.1 million cases (90% CrI 2.3–6.4) of foodborne gastroenteritis and 5,140 cases

(90% CrI 3,530–7,980) of nongastrointestinal foodborne illness occurred annually. Most foodborne illness occurs as gastroenteritis, but the effect of nongastrointestinal illnesses and sequelae are substantial because they can result in hospitalization and, occasionally, death. We identified that over time, the incidence of all foodborne gastroenteritis declined, but the incidences of salmonellosis and campylobacteriosis increased, although changes were not significant due to amount of uncertainty inherent in our estimates. These findings should assist policy makers to advocate for improved regulation and control of foodborne disease for specific pathogens.

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Foodborne Illness, Australia, Circa 2000 and Circa 2010

Technical Appendix 1

Data Sources

Estimates of illness based on surveillance data used notifiable surveillance data at the national or State level or other surveillance through the OzFoodNet Outbreak Register. Estimates of incidence were also calculated based on the 2008 Australian National Gastroenteritis Survey (NGSII) together with a fractional pathogen approach derived from cohort studies, such as the Water Quality Study (1–3). The data source and estimation approach used for each pathogen is explained in the Table.

Technical Appendix 1 Table. Data sources and estimation approach used for each pathogen or syndrome*

Pathogen or illness	Data Source	Estimation Approach
<i>Campylobacter</i> spp.	NNDSS	Notifiable Surveillance
<i>Salmonella</i> spp., nontyphoidal†	NNDSS	Notifiable Surveillance
<i>Salmonella enterica</i> serotype Typhi	NNDSS	Notifiable Surveillance
<i>Shigella</i> spp.	NNDSS	Notifiable Surveillance
<i>Cryptosporidium</i> spp.	NNDSS	Notifiable Surveillance
Hepatitis A	NNDSS	Notifiable Surveillance
<i>Listeria monocytogenes</i>	NNDSS	Notifiable Surveillance
<i>Giardia lamblia</i>	State Surveillance	Notifiable Surveillance
STEC	State Surveillance	Notifiable Surveillance
<i>Vibrio parahaemolyticus</i>	State Surveillance	Notifiable Surveillance
<i>Yersinia enterocolitica</i>	State Surveillance	Notifiable Surveillance
Other pathogenic <i>Escherichia coli</i>	NGSII (1) and WQS (2,3)	Pathogen Fraction
Adenovirus	NGSII (1) and WQS (2,3)	Pathogen Fraction
Astrovirus	NGSII (1) and WQS (2,3)	Pathogen Fraction
Norovirus	NGSII (1) and WQS (2,3)	Pathogen Fraction
Rotavirus	NGSII (1) and WQS (2,3)	Pathogen Fraction
Sapovirus	NGSII (1) and WQS (2,3)	Pathogen Fraction
<i>Bacillus cereus</i>	OzFoodNet Outbreak Register	Other Surveillance
<i>Clostridium perfringens</i>	OzFoodNet Outbreak Register	Other Surveillance
<i>Staphylococcus aureus</i>	OzFoodNet Outbreak Register	Other Surveillance
Ciguatera	OzFoodNet Outbreak Register	Other Surveillance
Scombrototoxicosis	OzFoodNet Outbreak Register	Other Surveillance
<i>Toxoplasma gondii</i>	U.S. Seroprevalence Study (4)	Special Calculations

*NGSII, National Gastroenteritis Survey II; NNDSS, National Notifiable Disease Surveillance System; STEC, Shiga toxin–producing *Escherichia coli*; WQS, Water Quality Study.

†Refers to nontyphoidal *Salmonella enterica* serotypes.

Notifiable Surveillance: National Notifiable Disease Surveillance Scheme and State Notifications

The Australian National Notifiable Disease Surveillance System (NNDSS) provides national data for pathogens that are notifiable in Australia, such as *Salmonella* spp., *Shigella* spp. and *Cryptosporidium* spp. Some pathogens are notifiable in some States, but not in others; for

example, *Campylobacter* spp. is not notifiable in New South Wales, but is notifiable in all other States. In these cases, we use notification data for the available States and included a population adjustment multiplier to estimate national notification rates (see online Technical Appendix 2, <http://wwwnc.cdc.gov/EID/article/20/11/13-1315-Techapp2.pdf>). In each case, we have used the total number of confirmed notifications for all available years over the period 2006–2010.

Additionally, we requested further data through the Communicable Disease Network of Australia (CDNA) to determine the proportion of cases that were domestically acquired in Australia. Details of the use of these data are described in online Technical Appendix 2 under the section title Domestically Acquired Multiplier.

Other Surveillance: OzFoodNet Outbreak Register

The OzFoodNet Outbreak Register includes all outbreaks identified over the period 2006–2008, providing data on the number of persons ill in each outbreak, the pathogen identified, and the total number of persons with laboratory confirmed illness in each outbreak.

National Gastroenteritis Survey II 2008

The NGSII was a nationally representative telephone survey conducted by the Department of Health and Ageing, the New South Wales Food Authority and the National Centre for Epidemiology and Population health in 2008–2009 to improve estimates of burden of gastroenteritis in Australia. It provides age-specific rates of gastroenteritis in the community.

Research Studies

We used Australian and international cohort studies to assess the proportion of gastroenteritis that is due to specific pathogens. A key study is the 1997 Water Quality Survey, which was a double-blinded, randomized, controlled trial of families conducted in Melbourne, Australia between September 1997 and February 1999 (2,3). Six hundred families were allocated to receive either real or sham water treatment units installed in their houses and study participants reported any gastroenteritis symptoms weekly. The study provides testing data on 795 fecal specimens identifying pathogens causing gastroenteritis, and we used this data to calculate a pathogen fraction multiplier for included pathogens (online Technical Appendix 2). As there was no significant difference in incidence of gastroenteritis in control and experimental families, the study found that waterborne pathogens do not play a major role in gastroenteritis in Melbourne (2).

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Foodborne Illness, Australia, Circa 2000 and Circa 2010

Technical Appendix 2

Calculating Community Incidence

Approaches and Distributions

We adopted three main approaches to calculating the incidence of illness in the community. These three approaches are based on the source of the data as

1. Notifiable surveillance approach using data from the National Notifiable Diseases Surveillance System (NNDSS) or State notifications;
2. Pathogen fraction approach using data from the 2008 National Gastroenteritis Survey II (NGSII) together with cohort studies, such as the Water Quality Study;
3. Other surveillance approach using data from the OzFoodNet Outbreak Register, or from hospitalizations

We considered these approaches to form a hierarchy, with the notifiable surveillance approach used by preference, and outbreak data used only when other sources were not available. For each approach, the final estimate was produced from a statistical model that incorporates uncertainty in case numbers and in multipliers using probability distributions. That is, at each stage of calculation, the estimate was represented by a probability distribution, and our final estimates and credible intervals were computed from these distributions. Where data for multiple approaches were available, we computed both and used the lower-hierarchy estimates as an informal cross-check.

Figures 1, 2, and 3 provide flowcharts explaining this approach. In each flowchart, the left-hand column provides a description of each input or output distribution, the central column provides a pictorial representation of the distribution, and the right-hand column describes the

type and source of data underlying each input distribution. In each case, input data arises from specific data sources (discussed in online Technical Appendix 1, <http://wwwnc.cdc.gov/EID/article/20/11/13-1315-Techapp1.pdf>), or from multipliers discussed below. We used three main input distribution types: empirical, PERT, and lognormal.

Empirical Distribution

Source distributions on the number of cases were typically represented by an empirical or discrete distribution driven by the data. For example, if the number of cases notified to NNDSS for the years 2006–2010 were 15416, 16980, 15539, 16075, and 16967, we would represent this as a discrete distribution with 20% of the probability mass at 15416, 20% of the probability mass at 16980, and so on. This use of empirical distributions for such data was used previously by Scallan et al. (1), and allowed us to avoid any assumptions about the expected shape of the distribution.

PERT Distribution

The PERT distribution is widely used for expert elicitation and risk assessment studies. It is based on the β distribution, and within the computer software @Risk, can be specified either using a minimum, maximum and modal value, or by three percentile points, such as a median value and 95% credible intervals. We used this distribution widely in our analysis, as it allows for asymmetric distributions, and can be easily produced from many data sources including expert elicitation

Lognormal Distribution

When re-calculating our underreporting multipliers we discovered that the PERT distribution did not adequately capture the shape of these multipliers. We adopted a lognormal distribution instead, as the distribution providing the best fit as measured by @Risk, and demonstrating an improved fit on the normal distribution used previously (2).

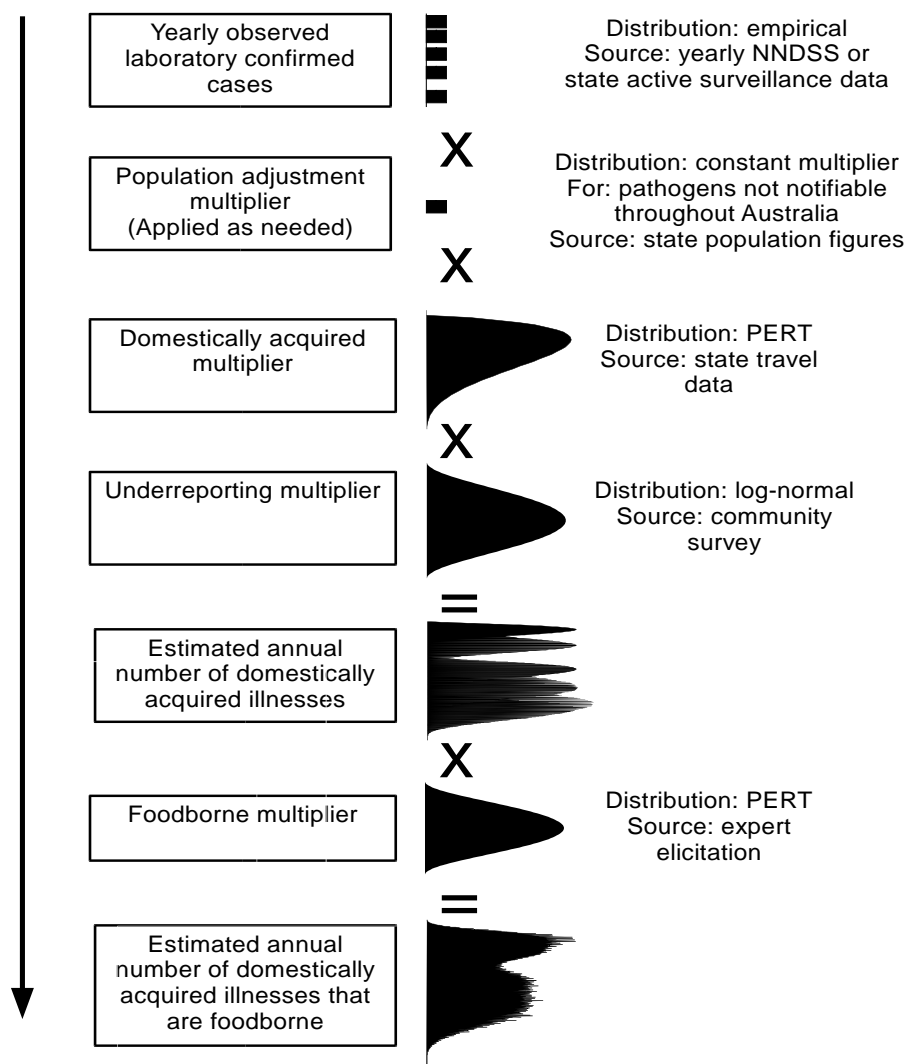
Multipliers

Figures 1, 2, and 3 give flowcharts for calculating foodborne disease illness using key multipliers either to scale up (surveillance approaches) from detected cases to the full community burden, or to scale down (pathogen fraction approach) from all gastroenteritis to the proportion that is due to specific pathogens.

Population Adjustment Multiplier

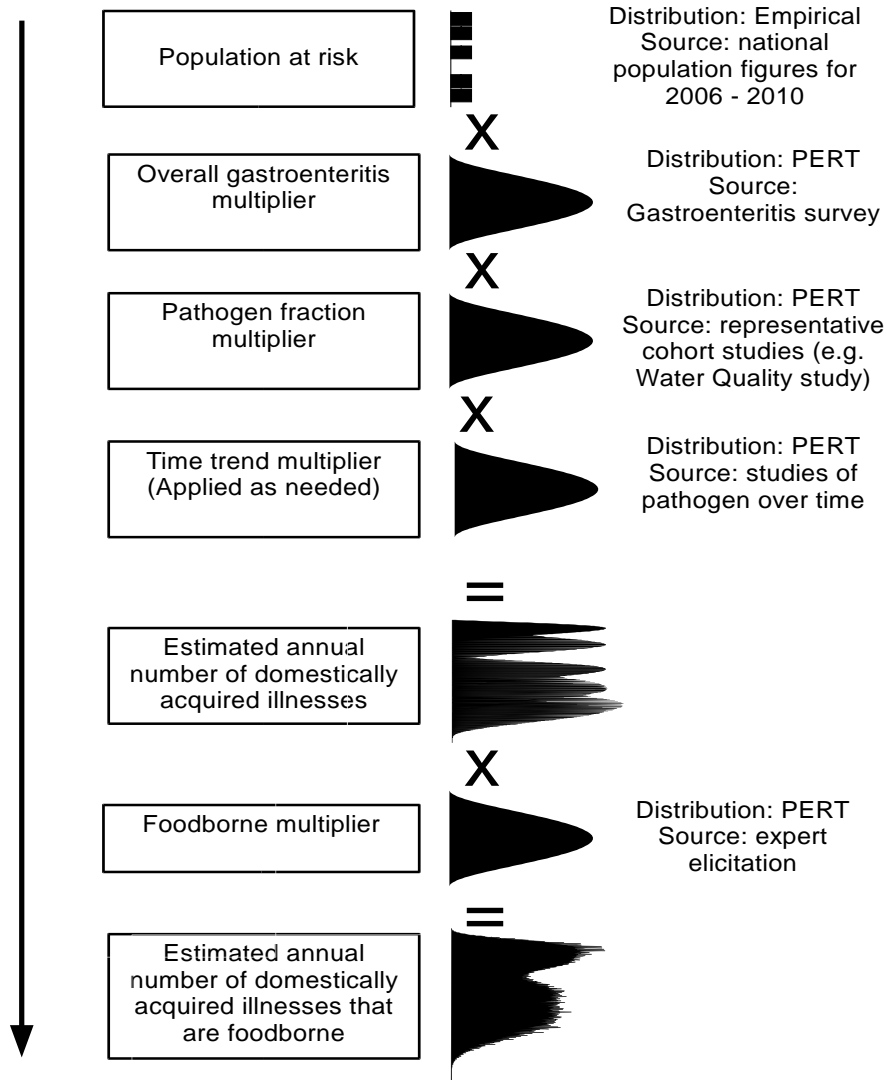
This multiplier was used where notifiable surveillance data are not available for all States in Australia, and was necessary to scale up the number of infections according to the proportion of the population covered by the surveillance data. For example, *Campylobacter* spp. is notifiable in all States except New South Wales. In this example we adjusted our total number of cases for the remaining States by a population adjustment multiplier of 1.5 to approximate the total number of cases we would expect to see if all States undertook notifiable surveillance.

Calculating the total number of cases using surveillance data



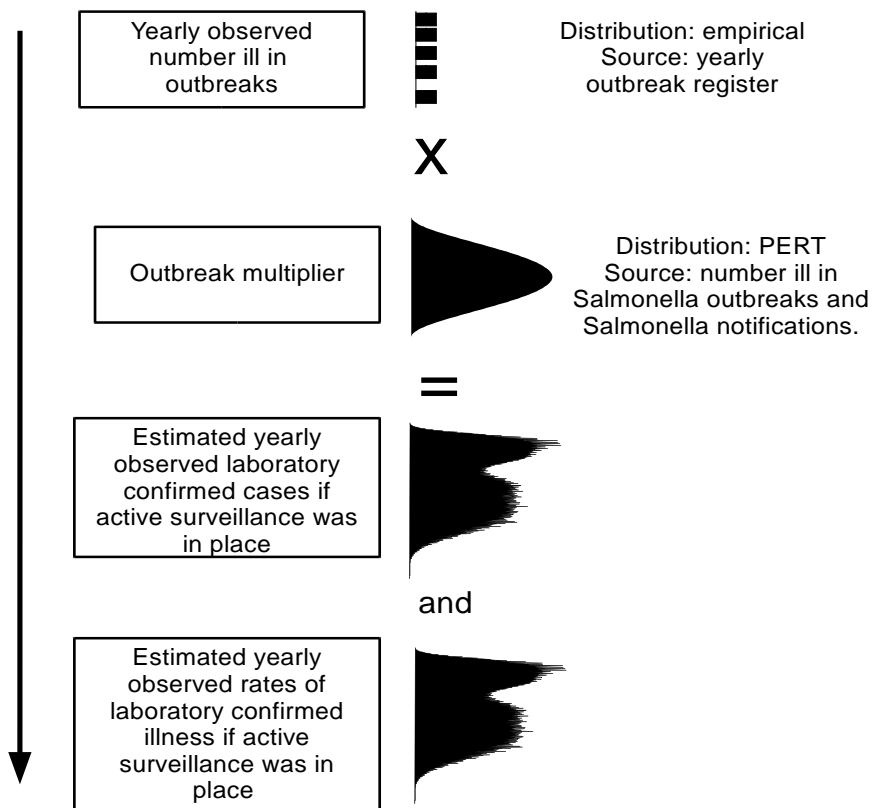
Technical Appendix 2 Figure 1. Flowchart for the notifiable surveillance approach used to calculate the estimated annual number of domestically acquired illnesses that are foodborne.

Calculating total number of cases by pathogen fraction approach



Technical Appendix 2 Figure 2. Flowchart for the pathogen fraction approach used to calculate the estimated annual number of domestically acquired illnesses that are foodborne.

Calculating rates and numbers of cases using outbreak data



Calculation of full rates and numbers of domestically acquired foodborne illness then proceeds as for surveillance flow charts.

Technical Appendix 2 Figure 3. Flowchart for the other surveillance approach used to calculate the estimated annual number of domestically acquired illnesses that are foodborne.

Domestically Acquired Multiplier

For some pathogens, a proportion of cases acquired their infections overseas. As data from the Water Quality Study used for the pathogen fraction calculations was centered on families, we assumed all these incident cases were domestically acquired. For *Campylobacter* spp., *Cryptosporidium* spp., hepatitis A, *Listeria monocytogenes*, nontyphoidal *Salmonella enterica* serotypes (hereafter referred to as nontyphoidal *Salmonella* spp.), *Salmonella enterica* serotype Typhi, *Shigella* spp., and Shiga toxin-producing *Escherichia coli* (STEC), the domestically acquired multiplier was calculated from NNDSS data on the proportion of cases that acquired their infection within Australia. This data contained several missing entries,

varying by pathogen, State and year, with the most complete data for Victoria and Western Australia. We considered four methods for adjusting for this missing data:

1. Extrapolate travel patterns from Western Australia to the Northern Territory and travel patterns from Victoria to all other States;
2. Extrapolate travel patterns from Western Australia to both the Northern Territory and Queensland, and travel patterns from Victoria to all other States;
3. Discard all missing data and calculate the proportion of cases acquired in Australia for the existing data only;
4. Assume all unidentified cases are domestically acquired

We adopted method 1 as the primary approach, and used the other methods as a comparison and to identify an uncertainty range for the multiplier. Specifically, the median estimate was made using all 5 years of data combined, while the minimum and maximum value reflects the largest and smallest proportion estimated by all four methods over each year of 2006–2010. Table 1 presents the resulting parameters for the PERT distribution, including median value, minimum and maximum, together with the estimations used by Hall et al. (3) for Australian estimates circa 2000. For *Cryptosporidium* spp., nontyphoidal *Salmonella* spp., and *Shigella* spp., estimates on the full data over 2006–2010 using methods 1 and 3 were reassuringly similar, while the expanded ranges reflect the yearly variability and sensitivity to missing data. Larger differences are seen for hepatitis A, *S. enterica* serotype Typhi, and STEC. There were very few missing data for hepatitis A and *S. enterica* serotype Typhi which raises our confidence in these estimates. Only 0 to 2 overseas cases of STEC were recorded per year, and this is reflected in the higher estimate of domestically acquired infection for this pathogen. This multiplier was also used for calculations of hospitalizations and deaths for other pathogenic *Escherichia coli*.

Estimates for the domestically acquired multiplier for *Giardia lamblia* were made using Victorian data over 2006–2009 (4–7), using the total proportion to derive the median and the variability over years to give a range. Domestically acquired multipliers for *Vibrio parahaemolyticus* and *Yersinia enterocolitica* were calculated from Western Australian data in a similar manner using OzFoodNet Annual Reports from 2006–2010. Given the higher rate of

overseas acquired infections in WA as compared with other jurisdictions, we reduced the proportion overseas for other States using a multiplier of 0.72 based on data for nontyphoidal *Salmonella* spp. Even with this adjustment, the multiplier for *V. parahaemolyticus* is much lower than that used in the U.S. suggesting a greater proportion of overseas-acquired cases in Australia (1); more information on the behavior of this pathogen in States outside Western Australia would be valuable to confirm our results.

Finally, we assumed that all cases of adenovirus, *Bacillus cereus*, ciguatera, *Clostridium perfringens*, *L. monocytogenes*, norovirus, rotavirus, scombrototoxicosis, *Staphylococcus aureus*, and *Toxoplasma gondii* were acquired in Australia. Domestically acquired multipliers were not needed for the remaining pathogens (astrovirus and sapovirus) for which incidence was calculated using the pathogen fraction approach, and that do not have specific codes to calculate hospitalizations and deaths.

Technical Appendix 2 Table 1. Estimated proportion of domestically acquired foodborne infections circa 2010 compared with previously published estimates for circa 2000, Australia*

Pathogen or Illness	Estimated % (range) of domestically acquired foodborne illnesses	
	Circa 2010	Circa 2000
Adenovirus	100 (100–100)	
<i>Bacillus cereus</i>	100 (100–100)	
<i>Campylobacter</i> spp.	97 (91–99)	96
Ciguatera	100 (100–100)	
<i>Clostridium perfringens</i>	100 (100–100)	
<i>Cryptosporidium</i> spp.	97 (92–99)	
<i>Giardia lamblia</i>	85 (84–89)	
Hepatitis A	58 (42–77)	
<i>Listeria monocytogenes</i>	100 (100–100)	
Norovirus	100 (100–100)	
Other pathogenic <i>Escherichia coli</i>	99 (93–100)	
Rotavirus	100 (100–100)	
<i>Salmonella</i> spp., nontyphoidal†	85 (70–95)	92
<i>Salmonella enterica</i> serotype Typhi	11 (2–25)	
Scombrototoxicosis	100 (100–100)	
<i>Shigella</i> spp.	70 (45–84)	60
<i>Staphylococcus aureus</i>	100 (100–100)	
STEC	99 (93–100)	79
<i>Toxoplasma gondii</i>	100 (100–100)	
<i>Vibrio parahaemolyticus</i>	18 (0–33)	
<i>Yersinia enterocolitica</i>	90 (80–100)	98

*Data circa 2000 was obtained for select pathogens and illnesses from 2 states (Victoria and South Australia) (8) Range was not provided. STEC, Shiga toxin-producing *Escherichia coli*.

†Refers to nontyphoidal *Salmonella enterica* serotypes.

Underreporting Multiplier

Only a fraction of community cases visit a health professional, have a sample taken and have their illness recorded in surveillance data. Using data from Hall et al. (2), we estimated underreporting multipliers based on lognormal distributions of 7 (95% Credible Interval 4–14) for nontyphoidal *Salmonella* spp., 10 (95% CrI 6.5–18.5) for *Campylobacter* spp., and 8 (95% CrI 3–18.5) for STEC. Where underreporting multipliers were needed for other pathogens, we

applied the nontyphoidal *Salmonella* spp. multiplier except in the case of pathogens leading to very severe illness (hepatitis A, *L. monocytogenes*, and *S. enterica* serotype Typhi) where the underreporting multiplier was assumed to be 2 (95% CrI 1–3). Details of the choice of multiplier for each pathogen are provided in online Technical Appendix 4 (<http://wwwnc.cdc.gov/EID/article/20/11/13-1315-Techapp4.pdf>).

Foodborne Multiplier

For most pathogens, we estimated the proportion of illness that is foodborne using data from Delphi based expert elicitations. For nine pathogens, we used a 2009 elicitation, and for another eight, we used a similar 2005 elicitation (9). The 2009 elicitation was informed by systematic reviews for each pathogen that included scientific literature, reports and surveillance data. Eleven experts estimated the proportion of illness transmitted via food through three rounds: the first round taking place after training questions, the second round after they had been provided with systematic reviews for all pathogens, and the final round after a 1-day workshop in which experts discussed each pathogen. At each step, experts were asked to estimate the proportion of transmission that is due to food, environment, water, animal or person-to-person transmission, making sure that these proportions summed to 1. The experts were then asked to give 90% certainty bounds for their foodborne proportion. Foodborne proportion estimates and intervals from the final stage of the elicitation were combined using PERT distributions. We extrapolated sapovirus from elicited norovirus estimates, and used best judgment assumptions for three additional viruses and the two marine biotoxins. See Table 2 for a listing of pathogens, multipliers and the data source for each. A comparison of these estimates with those used in prior studies is provided elsewhere (9).

Expert elicitation data from 2009 includes a best estimate and 90% interval for *Campylobacter* spp., *C. perfringens*, STEC, other pathogenic *E. coli*, nontyphoidal *Salmonella* spp., *Shigella* spp., norovirus, hepatitis A, and *L. monocytogenes*. We fitted a PERT distribution to each expert's assessment, fitting the best estimate as the median and setting the 90% interval where possible. In a few cases, we could not fit a PERT distribution in this way, and either had to adjust the best estimate to be the mode of the distribution (if the median point was too close to an upper or lower bound), or adjust an interval bound to be a min or max if the PERT distribution led to values outside the interval 0 to 1. A combined empirical distribution was

calculated by computing the point-wise mean value of the individual uncertainty distribution for each expert. The median, 5% and 95% percentiles of this empirical distribution were then used to describe a final PERT distribution that was input into the relevant @Risk spreadsheet.

The 2005 questionnaire provided a best estimate from participants. To include uncertainty in this estimate, we generated a 90% credible interval about each estimate, assuming an upper bound 10 percentage points higher and a lower bound 10 percentage points lower. For example, an estimate of 30% foodborne was modeled as a PERT distribution with median as 0.3, 95% bound 0.4, and 5% bound 0.2. The exception to this was where estimates were too close to zero (or one) for this method. We then assumed symmetric estimates half the distance from zero (or one). That is, an estimate of 5% foodborne was modeled as a PERT with median as 0.05, 5% bound as 0.025 and 95% bound as 0.075. The combined distribution was calculated as for the expert elicitation data. The 2005 elicitation did not achieve consensus for some pathogens; in particular, best estimates ranged from 2%–95% for *S. enterica* serotype Typhi, 5%–100% for *V. parahemolyticus*, and 33%–90% for *Y. enterocolitica*. Given the variability arising from these expert data, we tested the sensitivity of our results to the choice of distribution by simulating the full empirical distribution of the foodborne multiplier for each of these pathogens, and compared estimates of foodborne illness with those using the PERT distribution. In general, median estimates were little changed, but credible intervals were a little wider under the empirical distribution. The largest change was for *Y. enterocolitica*, where the estimate of domestically acquired foodborne illness was 1,150 (650–1950) using a PERT distribution, and 1,100 (350–2,050) using the empirical distribution.

Outbreak Multiplier

For pathogens that are not captured by notifiable surveillance or by cohort studies, we used data from outbreaks in the other surveillance approach. Only a fraction of cases are associated with outbreaks. The outbreak multiplier adjusts for this to estimate the total number of cases that would be captured if notifiable surveillance was in place for that pathogen. Many of the pathogens for which this method was used have a short duration of illness, and thus low rates of laboratory confirmation. To adjust for this, we calculated the multiplier based on total number of ill (but not necessarily lab confirmed) cases associated with a confirmed outbreak (where laboratory confirmation of at least one case or of a food source has been occurred). We chose to

use nontyphoidal *Salmonella* as the reference pathogen for the outbreak multiplier as it has the most complete data. The outbreak multiplier was calculated as the ratio of the number of ill cases in outbreaks of nontyphoidal *Salmonella* spp. to the total number of laboratory confirmed domestically acquired cases of nontyphoidal *Salmonella* spp. in the NNDSS for the same year. For example, in 2008 there were 8, 316 laboratory confirmed cases of nontyphoidal *Salmonella* spp. in NNDSS, of which 85% (range: 70–90) were assumed to be acquired in Australia. The total number of ill cases associated with nontyphoidal *Salmonella* spp. outbreaks in 2008 was 524, giving an outbreak multiplier of around 13.5 for this year. Extending this approach to calculate multipliers for each year from 2006–2008, and for data for all years combined, we estimate an outbreak multiplier of 14, with range 5–20.

Gastroenteritis Multiplier

For pathogens captured by cohort studies such as the Water Quality Study (10,11), we attributed a proportion of all gastroenteritis cases to that pathogen using the pathogen fraction approach (see Figure 2). The first step of this approach was to determine the total incidence of gastroenteritis. To do this we used the NGSII study to estimate the total number of gastroenteritis episodes per person per year, weighted by the Australian population. This estimate served to provide a gastroenteritis multiplier, which was then multiplied by the total Australian population for the years 2006–2010 to give the estimated number of cases of gastroenteritis for each year. The gastroenteritis multiplier was modeled as an alternative PERT distribution with median 0.74 and 95% interval (0.64–0.84), based on the estimates and uncertainty intervals estimated by the NGSII study.

Technical Appendix 2 Table 2. Estimates of the foodborne multiplier with 90% credible interval using PERT distributions for each of the 23 pathogens*

Pathogen or Illness	Foodborne multiplier (90% CrI)†	Data source‡
Adenovirus	0.02 (0.01–0.03)	Assumption
Astrovirus	0.02 (0.01–0.03)	Assumption
<i>Bacillus cereus</i>	1.00 (0.98–1.00)	2005 EE as PERT
<i>Campylobacter</i> spp.	0.77 (0.62–0.89)	2009 EE as PERT
Ciguatera	1.00 (1.00–1.00)	Assumption
<i>Clostridium perfringens</i>	0.98 (0.86–1.0)	2009 EE as PERT
<i>Cryptosporidium</i> spp.	0.10 (0.01–0.27)	2005 EE as PERT
Other pathogenic <i>Escherichia coli</i>	0.23 (0.08–0.55)	2009 EE as PERT
<i>Giardia lamblia</i>	0.06 (0.01–0.50)	2005 EE as PERT
Hepatitis A	0.12 (0.05–0.24)	2009 EE as PERT
<i>Listeria monocytogenes</i>	0.98 (0.90–1.00)	2009 EE as PERT
Norovirus	0.18 (0.05–0.35)	2009 EE as PERT
Rotavirus	0.02 (0.01–0.03)	Assumption
<i>Salmonella</i> spp., nontyphoidal§	0.72 (0.53–0.86)	2009 EE as PERT
<i>Salmonella enterica</i> serotype Typhi	0.75 (0.02–0.97)	2005 EE as PERT
Sapovirus	0.18 (0.05, 0.35)	Norovirus multiplier
Scombrototoxicosis	1.00 (1.00, 1.00)	Assumption
<i>Shigella</i> spp.	0.12 (0.05, 0.23)	2009 EE as PERT

Pathogen or Illness	Foodborne multiplier (90% CrI) [†]	Data source [‡]
<i>Staphylococcus aureus</i>	1.00 (0.95, 1.00)	2005 EE as PERT
STEC	0.56 (0.32, 0.83)	2009 EE as PERT
<i>Toxoplasma gondii</i>	0.31 (0.04, 0.74)	2005 EE as PERT
<i>Vibrio parahaemolyticus</i>	0.75 (0.05, 0.96)	2005 EE as PERT
<i>Yersinia enterocolitica</i>	0.84 (0.28, 0.94)	2005 EE as PERT

*Program evaluation review technique (PERT) is a commonly used distribution in expert elicitation and is based on a two parameter Beta distribution. STEC, Shiga toxin–producing *Escherichia coli*.

[†]Credible Intervals.

[‡] See Vally et al (8) for a comparison of these estimates with those used in prior studies. EE = Expert elicitation.

[§]Refers to nontyphoidal *Salmonella enterica* serotypes.

Pathogen Fraction Multiplier

The pathogen fraction multiplier attributed a proportion of the total number of gastroenteritis episodes to particular pathogens. Our primary data source for this was the Water Quality Study (10,11). While we also used data from the UK IID2 study (12) as a comparator, we found the Water Quality study gave the most reliable picture of the burden of illness due to different pathogens in Australia. The data from the study were age-adjusted (using age ranges 0–4, 5–14, 15+) to the Australian population (circa 2010) to take account of the higher numbers of children in the Water Quality study. For example, the raw data for adenovirus in the Water Quality study was 9 positive samples from a total of 713 samples taken from participants with a highly credible episode of gastroenteritis. However, 8 of those positives were from participants aged 0–4 years old, an age group over sampled in the study. Using data on the incidence of gastroenteritis by age from the NSGII study, and the Australian population as a reference, we calculated age-adjusted estimates for each pathogen based on the Water Quality Study data. For example, for adenovirus, we derived an estimate of 4 samples positive for adenovirus from 713 gastroenteritis episodes. This gave us a pathogen fraction multiplier of 0.0056 (95% CI: 0.0015–0.0143), which was then modeled in @Risk using an alternative PERT distribution. Note that the pathogen sheets provided in online Technical Appendix 4 provide the age adjusted estimates for each pathogen, so will differ slightly from studies reporting findings of the Water Quality Study.

Finally, we could not find any Australian cohort study that gave estimates of prevalence of astrovirus or sapovirus for all age groups. Instead, we used pathogen fraction multiplier from the Water Quality Study for adenovirus and norovirus, together with cohort data from children (13) to calculate multipliers relating astrovirus to adenovirus, and sapovirus to norovirus (14). Although the use of children only in this approach is not ideal, it allowed us to use Australian data. We also considered an alternative approach using data from the UK infectious intestinal disease study 2 (IID2) (12), but found this led to unexpectedly high estimates for astrovirus and sapovirus that were not consistent with estimates for other viral pathogens estimated using data

from the Water Quality Study (10,11). These differences perhaps arise from differences in the gastroenteritis case definitions in the UK IID2 study (12) and our Australian NGSII study.

Time Trend Multiplier

The Water Quality Study (10,11) was undertaken before the addition of a rotavirus vaccine to the Australian vaccination schedule in 2007. In calculating rotavirus incidence circa 2010, we included a time-trend multiplier to adjust for the reduction in rotavirus in 2010 compared with pre-vaccination levels. In calculating this multiplier, we used data from a study of rotavirus hospitalizations by age before and after the introduction of the vaccination program (15). By comparing age-specific hospitalization rates in 2010 with that before vaccination, we were able to estimate a time-trend multiplier of 0.34 (95% Confidence Interval 0.32–0.36) to adjust for the decline in rotavirus following vaccination.

Toxoplasmosis – Special Calculations

The calculations for toxoplasmosis differed from all other methods, as we used U.S. seroprevalence studies to estimate yearly incident cases assuming a constant force of infection with age (16). While there is an Australian study of toxoplasmosis (17), we felt the sample size was too small to rely on for this estimate. In adopting this U.S. study rather than European studies (see Pappas et al. (18) for a systematic review), we ensure comparability with our prior work, and take a conservative approach to estimating Australian incidence of toxoplasmosis. We then adjusted this incidence estimate by a “proportion symptomatic” multiplier of 15% (90% CrI 11–21) in line with the approach used by Hall et al circa 2000 (3) and that of Scallan et al. (1).

Comparison with estimates from 2000

Several multipliers used in these calculations have changed since our study circa 2000 (3). These changes do not reflect altered behavior of pathogens, but rather new knowledge and better estimates of the multipliers involved. Owing to these changed multipliers, a direct comparison of this study with that circa 2000 is misleading. To provide a more appropriate comparison, we have recalculated all estimates for 2000 using new multipliers. Our aim here is to remove components of the time comparison that we know to be misleading. As for 2010, all estimates for 2000 include all uncertainty due to (new) multipliers.

Unknown Pathogens

We used the NGSII survey of gastroenteritis conducted in 2008–2009 to estimate the total envelope of domestically acquired gastrointestinal illness, and so calculated the incidence of unknown pathogens by subtracting the incidence of known pathogens causing domestically acquired gastrointestinal pathogens from that of the survey. Credible intervals were estimated using @Risk, assuming all cases in the NGSII were domestically acquired. We calculated the foodborne multiplier for all known pathogens of 25% (90% CrI: 15–39) as a weighted average of the foodborne multiplier for each pathogen, weighted by the number of domestically acquired cases of each pathogen. Although this value is remarkably similar to that estimated by Scallan et al (1,19), it is worth noting that it is based entirely on Australian expert elicitation data, together with incidence calculations using Australian data, and so is entirely independent of that study. Examination of the two studies will identify differences in many components of the calculations. The foodborne multiplier was applied to unknown pathogens to estimate the total number of domestically acquired foodborne illness due to unknown pathogens, again using @Risk for credible intervals.

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Foodborne Illness, Australia, Circa 2000 and Circa 2010

Technical Appendix 3

Methods to Estimate Hospitalizations and Deaths

Data Sources

We used hospitalization data from all Australian States and Territories for 2006–2010 (where available), and deaths data from the Australian Bureau of Statistics, using ICD 10 codes for deaths and ICD 10AM codes for hospitalizations as in Table 1. Both astrovirus and sapovirus were excluded from this analysis as lacking appropriate codes in our data. Patients were included as a hospitalization if the appropriate code was included as the principal or an additional diagnosis. Table 2 shows the percentage of all hospital diagnoses that were listed as the principal diagnosis for each pathogen for 2010 (the year with most complete data). In our previous study (*1*), we used only data on principal diagnoses, with a multiplier of 2 (credible interval [CrI] 1–3) for all pathogens to model both principal and additional diagnoses. It is clear from Table 2 that diagnosis patterns vary considerably by pathogen, so that use of both principal and additional diagnosis data provides a more complete picture of hospitalizations.

Since we only had 1 year of hospitalization data for Victoria and 2 years for New South Wales, we had to extrapolate from these data to the remaining years to derive a distribution of the number of hospitalizations across all states, which was modeled as an empirical distribution. In most cases, we assumed the same number of hospitalizations each year, but some pathogens required further adjustment due to evident outbreaks or trends. For example, an outbreak of hepatitis A associated with sundried tomatoes coincided with the 1 year of hospitalization data for Victoria. We used a ratio of hospitalizations in South Australia to Victoria to estimate Victorian hospitalizations for the missing years. As vaccination against rotavirus resulted in a decrease in incidence, hospitalizations, and deaths, we used data post universal vaccination, from 2008–2010 only, to estimate hospitalizations circa 2010.

Approaches

To calculate estimates of hospitalizations and deaths, we used a statistical model that incorporates uncertainty in case numbers and in multipliers using probability distributions. That is, at each stage of the calculation, the estimate was represented by a probability distribution, and our final estimates and CrIs were computed from this distribution. Figures 1 and 2 provide flowcharts of the approach for hospitalizations, where the left-hand column gives a description of the input or output distribution, the central column provides a representation of the distribution, and the right-hand column describes the type and source of data underlying each input distribution. Input data was obtained from specific data sources (discussed above) or from multipliers that are described below. A fuller description of these probability distributions is provided in the methods section for incidence.

Multipliers

Underdiagnosis Multiplier

Recorded hospitalizations and deaths associated with each pathogen reflect only those individuals that have been tested and confirmed for the pathogen. Following previous studies, we adjusted for this using an underdiagnosis multiplier of 2 (1), including a distribution for the multiplier with range 1–3 as in Hall et al. (2) and Scallan et al. (3). We confirmed the appropriateness of the multiplier for hospitalizations as follows. First, we used the OzFoodNet Outbreak Register to calculate the proportion of all ill cases associated with an outbreak that were hospitalized. We then compared this proportion to the ratio of incidence to hospitalizations both with and without the underdiagnosis multiplier. Although there was some variability by pathogen, overall, we found that 3% of ill cases in the OzFoodNet Outbreak Register were hospitalized. In contrast, the ratio of all incident cases to all hospitalized cases was around 0.01 when the underdiagnosis multiplier was included (and 0.005 otherwise). Although outbreak cases may be more severe than all incident cases (on average), and under-ascertainment of cases or under-recording of hospitalizations may have biased our validation of the multiplier, our results suggest that an underdiagnosis multiplier is appropriate. Further work would assist in better quantifying this multiplier.

Domestically Acquired Multiplier

This multiplier adjusted for the proportion of cases that acquired infection in Australia, and was adopted from the method for incidence. More details of the data and methods behind this multiplier are provided in online Technical Appendix 2 (<http://wwwnc.cdc.gov/EID/article/20/11/13-1315-Techapp2.pdf>).

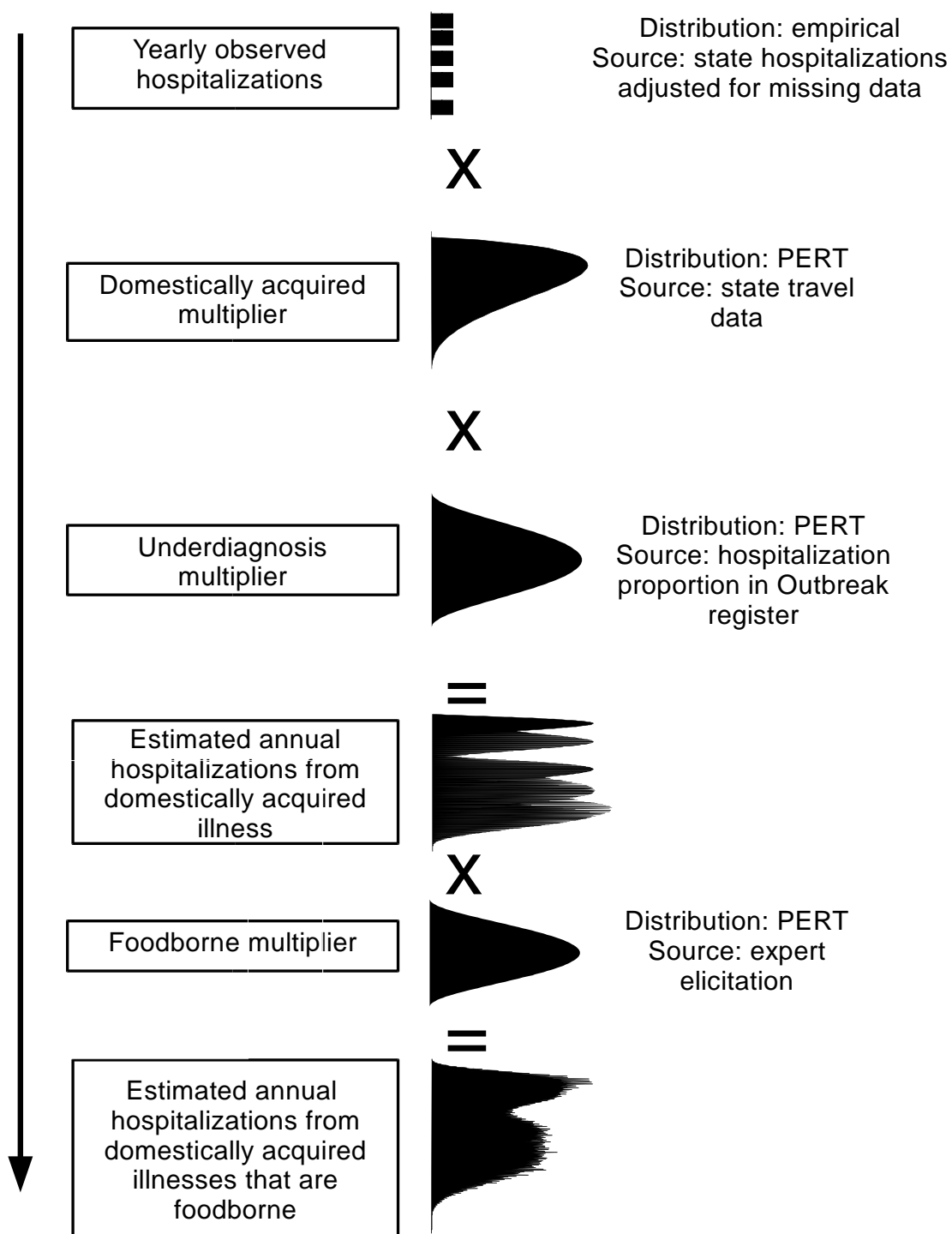
Foodborne Multiplier

This multiplier adjusted for the proportion of illness that is foodborne using expert elicitation data, and was used for incidence, hospitalizations and deaths. More details are provided in online Technical Appendix 2.

Hospitalizations and Deaths Due to Unknown Pathogens

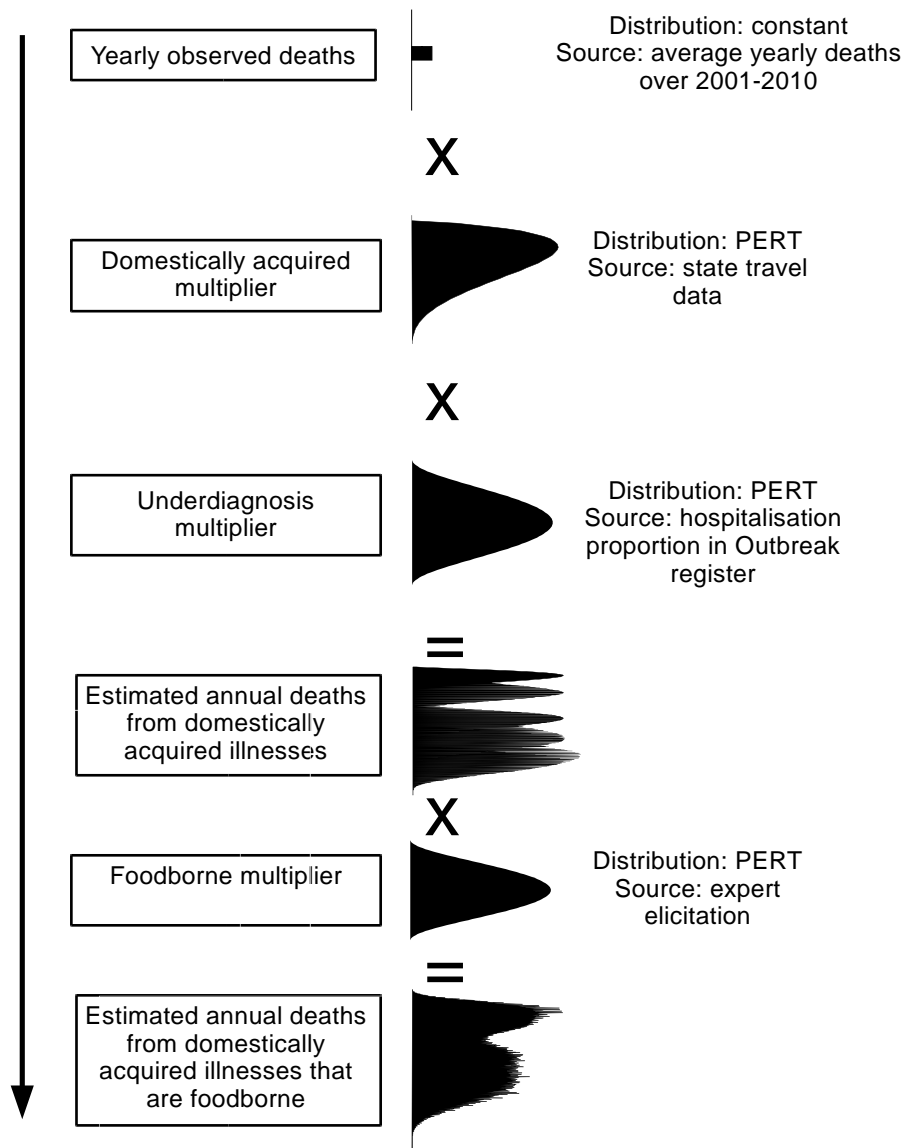
A large proportion of hospitalizations and deaths did not identify the source of infection (see “other” codes in Table 1). These data were adjusted and reported as follows for hospitalizations, with a similar approach used for deaths. First, the total number of hospitalizations due to unknown pathogens was calculated from the appropriate codes. We then subtracted from this number the hospitalizations that were attributed to known pathogens according to the underdiagnosis multiplier described above. That is, where total numbers of known gastrointestinal pathogens were increased to adjust for underdiagnosis, this increase was subtracted from the total unknown gastrointestinal pathogens. We assumed a domestically acquired multiplier of 1 for unknown pathogens, but adjusted for the foodborne multiplier using an average over known pathogens, weighted by the number of hospitalizations for each pathogen. For hospitalization data, this gave a foodborne multiplier of 44% (90% CrI 38–50), and for death data, a foodborne multiplier of 51% (90% CrI 36–71). Although Scallan et al. (3) do not report their weighted foodborne multipliers for hospitalizations and deaths, analysis of their tables suggest their values are 24% for hospitalizations and 52% for deaths. As noted in online Technical Appendix 2, our calculations are entirely independent; our hospitalization estimate is considerably higher although the estimate for deaths shows good agreement.

Calculating the total number of hospitalized cases



Technical Appendix 3 Figure 1. Flowchart for the approach used to calculate the estimated annual number of hospitalizations.

Calculating the total number of deaths



Technical Appendix 3 Figure 2. Flowchart for the approach used to calculate the estimated annual number of deaths.

Technical Appendix 3 Table 1. Mortality and Hospitalization codes for each pathogen*

Pathogen or Illness	Mortality ICD 10 Code and description	ICD 10AM
Adenovirus	A08.2: Adenoviral enteritis	A08.2: Adenoviral enteritis
<i>Bacillus cereus</i>	A05.4: Foodborne <i>Bacillus cereus</i> intoxication	A05.4: Foodborne <i>Bacillus cereus</i> intoxication
<i>Campylobacter</i> spp.	A04.5: <i>Campylobacter</i> enteritis	A04.5: <i>Campylobacter</i> enteritis
Ciguatera	T61.0: Ciguatera fish poisoning	T61.0: Ciguatera fish poisoning
<i>Clostridium perfringens</i>	A05.2: Foodborne <i>Clostridium perfringens</i> intoxication	A05.2: Foodborne <i>Clostridium perfringens</i> intoxication
<i>Cryptosporidium</i> spp.	A07.2: Cryptosporidiosis	A07.2: Cryptosporidiosis
Guillain-Barré Syndrome	G61.0: Guillain-Barré syndrome	G61.0: Guillain-Barré syndrome
<i>Giardia lamblia</i>	A07.1: Giardiasis [lambliasis]	A07.1: Giardiasis [lambliasis]
Hepatitis A	B15: Acute hepatitis A	B15.9: Hepatitis A without hepatic coma

Pathogen or Illness	Mortality ICD 10 Code and description	ICD 10AM
Hemolytic-uremic syndrome	D59.3: Hemolytic-uremic syndrome	D59.3: Hemolytic-uremic syndrome
Irritable bowel Syndrome	K58: Irritable bowel syndrome	K58.0: Irritable bowel with diarrhea K58.9: Irritable bowel without diarrhea
<i>Listeria monocytogenes</i>	A32: Listeriosis	A32.0-A32.9: Listeriosis
Norovirus	A08.1: Acute gastroenteropathy due to Norwalk agent	A08.1: Acute gastroenteropathy due to Norwalk agent
Other pathogenic <i>Escherichia coli</i>	A04.0: Enteropathogenic <i>Escherichia coli</i> infection A04.1: Enterotoxigenic <i>Escherichia coli</i> infection A04.2: Enteroinvasive <i>Escherichia coli</i> infection A04.4: Other intestinal <i>Escherichia coli</i> infection	A04.0: Enteropathogenic <i>Escherichia coli</i> infection A04.1: Enterotoxigenic <i>Escherichia coli</i> infection A04.2: Enteroinvasive <i>Escherichia coli</i> infection A04.4: Other intestinal <i>Escherichia coli</i> infections
Reactive arthritis	M02.1: Postdysenteric arthropathy M02.8: Other reactive arthropathies	M02.1: Postdysenteric arthropathy, multiple sites M02.3: Reiter's disease, multiple sites M02.8: Other reactive arthropathies, multiple sites M03.2: Other postinfectious arthropathies in diseases classified elsewhere, multiple sites
Rotavirus	A08.0: Rotaviral enteritis	A08.0: Rotaviral enteritis
<i>Salmonella</i> spp., nontyphoidal†	A02: other <i>Salmonella</i> infections	A02.0-A02.9: Salmonellosis
<i>Salmonella enterica</i> serotype Typhi	A01: Typhoid and paratyphoid fevers	A01: Typhoid fever
Scombrototoxicosis	T61.1: Scombroid fish poisoning	T61.6: Scombroid fish poisoning
<i>Shigella</i> spp.	A03: Shigellosis	A03.0-A03.9: Shigellosis
<i>Staphylococcus aureus</i>	A5.0: Foodborne staphylococcal intoxication	A05.0: Foodborne staphylococcal intoxication
STEC	A04.3: Enterohemorrhagic <i>Escherichia coli</i> infection	A04.3: Enterohemorrhagic <i>Escherichia coli</i> infection
<i>Toxoplasma gondii</i>	B58: Toxoplasmosis	B58.0-B58.9: Toxoplasmosis
<i>Vibrio parahaemolyticus</i>	A05.3: Foodborne <i>Vibrio parahaemolyticus</i> intoxication	A05.3: Foodborne <i>Vibrio parahaemolyticus</i> intoxication
<i>Yersinia enterocolitica</i>	A04.6: Enteritis due to <i>Yersinia enterocolitica</i>	A04.6: Enteritis due to <i>Yersinia enterocolitica</i>
Other	A04.8: Other specified bacterial intestinal infection A04.9: Bacterial intestinal infection unspecified A05.8: Other specified bacterial foodborne intoxications A05.9: Bacterial foodborne intoxication unspecified A07.8: Other specified protozoa intestinal diseases A07.9: Protozoa intestinal disease, unspecified A08.3: Other viral enteritis A08.4: Viral intestinal infection, unspecified A09: Diarrhea and gastroenteritis of presumed infectious origin T61.2 Other fish and shellfish poisoning T61.8 Toxic effect of other seafood T61.9 Toxic effect of unspecified seafood T62: Toxic effect of other noxious substances eaten as food T64: Toxic effect of aflatoxin and other mycotoxin food contaminants	A08.4: Viral intestinal infection, unspecified A09: Diarrhea and gastroenteritis of presumed infectious origin A09.0: Other gastroenteritis and colitis of infectious origin A09.9: Other gastroenteritis and colitis of unspecified origin

*STEC, Shiga toxin-producing *Escherichia coli*.

†Refers to nontyphoidal *Salmonella enterica* serotypes.

Technical Appendix 3 Table 2. The percentage of all hospital diagnoses that were listed as principal for each pathogen, based on 2010 data for all States*

Pathogen or Illness	Percentage of all diagnoses listed as principal
Adenovirus	82
<i>Bacillus cereus</i>	75
<i>Campylobacter</i> spp.	79
Ciguatera	83
<i>Clostridium perfringens</i>	100
<i>Cryptosporidium</i> spp.	59

Pathogen or Illness	Percentage of all diagnoses listed as principal
Other pathogenic <i>Escherichia coli</i>	59
<i>Giardia lamblia</i>	34
Guillain-Barré syndrome	71
Irritable bowel syndrome	69
Hemolytic uremic syndrome	30
Hepatitis A	77
<i>Listeria monocytogenes</i>	48
Norovirus	37
Reactive arthritis	50
Rotavirus	77
<i>Salmonella</i> spp., nontyphoidal†	77
<i>Salmonella enterica</i> serotype Typhi	93
Scombrototoxicosis	100
<i>Shigella</i> spp.	76
<i>Staphylococcus aureus</i>	100
STEC	59
<i>Toxoplasma gondii</i>	39
<i>Vibrio parahaemolyticus</i>	50
<i>Yersinia enterocolitica</i>	64

*STEC, Shiga toxin–producing *Escherichia coli*.

†Refers to nontyphoidal *Salmonella enterica* serotypes.

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Foodborne Illness, Australia, Circa 2000 and Circa 2010

Technical Appendix 4

Pathogen and Illness Sheets

Adenovirus

Technical Appendix 4 Table 1. Primary Data: Water Quality Study; Alternate Data: IID2*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness:		
Gastroenteritis multiplier—based on the 2008 National Gastroenteritis Survey	Alternate PERT	2.5%, median, 97.5% values: 0.64, 0.74, 0.84
Pathogen fraction multiplier—based on age adjusted water quality study of an estimated 4 positive isolates per 713 specimens, (Hellard et al. (1))	Alternate PERT	2.5%, median, 97.5% values: 0.0015, 0.0056, 0.0143
Population adjustment:	Empirical	By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)		
Domestically acquired multiplier:		NA
All illnesses in the Water Quality Study were domestically acquired		
Time trend multiplier:		NA
No time trend		
Underreporting:		NA
Water Quality Study is community surveillance		
Total illness:	Outcome	5%, median, 95% values: 28800, 88400, 205000
Population at risk x gastroenteritis multiplier x pathogen fraction multiplier x time trend multiplier		
Rate of total illness per million:	Outcome	5%, median, 95% values: 1300, 4150, 9675
Circa 2010		
Foodborne multiplier:	Alternate PERT	5%, median, 95% values: 0.01, 0.02, 0.03
Assumed to be the same as rotavirus		
Total foodborne illness:	Outcome	5%, median, 95% values: 500, 1650, 4650
Total illness x foodborne multiplier		
Rate of foodborne illness per million:	Outcome	5%, median, 95% values: 25, 80, 215
Circa 2010		

*Longitudinal study of infectious intestinal disease in the UK. NA, not applicable.

Astrovirus

Technical Appendix 4 Table 2. Primary Data: Water Quality Study; Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness:		
Gastroenteritis multiplier—based on the 2008 National Gastroenteritis Survey	Alternate PERT	2.5%, median, 97.5% values: 0.64, 0.74, 0.84
Pathogen fraction multiplier—based on age adjusted water quality study of an estimated 4 positive isolates per 713 specimens, (Hellard et al. (1))	Alternate PERT	2.5%, median, 97.5% values: 0.0015, 0.0056, 0.0143
Pathogen comparison multiplier - Kirkwood multiplier (2) comparing adenovirus to astrovirus	Constant	0.76
Population adjustment:	Empirical	By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)		
Domestically acquired multiplier:		NA
All illnesses in the Water Quality Study were domestically acquired		
Time trend multiplier:		NA
No time trend		
Underreporting:		NA
Water Quality Study is community surveillance		
Total illness:	Outcome	5%, median, 95% values: 20900, 67100, 15500
Population at risk x gastroenteritis multiplier x pathogen fraction multiplier x time trend multiplier		
Rate of total illness per million:	Outcome	5%, median, 95% values: 1000, 3150, 7250
Circa 2010		
Foodborne multiplier:	Alternate PERT	5%, median, 95% values: 0.01, 0.02, 0.03
Assumed to be the same as rotavirus		
Total foodborne illness:	Outcome	5%, median, 95% values: 350, 1300, 3400
Total illness x foodborne multiplier		
Rate of foodborne illness per million:	Outcome	5%, median, 95% values: 20, 60, 160
Circa 2010		

*NA, not applicable.

Bacillus cereus

Technical Appendix 4 Table 3. Primary Data: Outbreak; Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness:	Empirical	By year (2006–2008): 14, 35, 75
The number of <i>B. cereus</i> outbreak-associated illnesses reported to OzFoodNet 2006–2008		
Population adjustment:	Empirical	By year (2006–2008): 20697880, 21015936, 21384427
Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)		
Domestically acquired multiplier:	PERT	Minimum, modal, maximum values: 1, 1, 1
Assumed to be 100% domestically acquired due to the short incubation period		
Underreporting:		

Model Input, Source and Comments	Distribution	Data for Model Input
Outbreak multiplier used to adjust from outbreak to surveillance (O-S)	PERT	Minimum, modal, maximum values: 5, 14, 20
Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> multiplier adapted from Hall et al. (3)	Log Normal	Mean, standard deviation: 7.44, 2.38
Total illness: Outbreak cases x Underreporting(O-S)(S-C) x Proportion travel-related	Outcome	5%, median, 95% values: 900, 3350, 10100
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 40, 150, 485
Foodborne multiplier: Based on 2005 expert elicitation	PERT	Minimum, modal, maximum values: 0.98, 1, 1
Total foodborne illness: Total illness x Foodborne multiplier	Outcome	5%, median, 95% values: 2900, 3350, 10100
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 95% values: 40, 150, 485

*NA, not applicable.

***Campylobacter* spp.**

Technical Appendix 4 Table 4. Primary Data: National Notifiable Disease Surveillance System (NNDSS); Alternate Data: Water Quality Study

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: NNDSS data. Available from: http://www9.health.gov.au/cda/source/rpt_4.cfm (Cited 2013 Nov 12)	Empirical	By year (1996–2000): 12169, 11984, 12647, 12373, 13676 By year (2006–2010): 15416, 16980, 15539, 16075, 16967
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (1996–2000): 18310714, 18517564, 18711271, 18925855, 19153380 By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Correction factor: <i>Campylobacter</i> spp. is not notifiable in New South Wales—based on Hall et al (3)	Constant	1.5
Domestically acquired multiplier: NNDSS travel data	PERT	Minimum, modal, maximum values: 0.91, 0.97, 0.99
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C). <i>Campylobacter</i> spp. multiplier adapted from Hall et al. (3)	Log Normal	Mean, standard deviation: 10.45, 2.98
Total illness: Reported cases (NNDSS) x travel adjustment x underreporting (S-C)	Outcome	5%, median, 95% values: 147000, 234000, 374000
Rate of total illness per million: circa 2010	Outcome	5%, median, 95% values: 6850, 10950, 17415
Foodborne multiplier: Expert elicitation study 2009	Alternate PERT	5%, median, 95% values: 0.62, 0.77, 0.89
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 1108500, 179000, 290000 (circa 2010) 5%, median, 95% values: 82500, 139000, 227000 (circa 2000)
Rate of foodborne illness per million: Circa 2010 and circa 2000	Outcome	5%, median, 9% values: 5050, 8400, 13650 (circa 2010) 5%, median, 9% values: 4500, 7400, 12200 (circa 2000)

Ciguatera

Technical Appendix 4 Table 5. Primary Data: Queensland Notifications; Alternate Data: Outbreak

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: The number of ciguatera notifications reported in Queensland in OzFoodNet Queensland Annual Reports 2006–2010	Empirical	By year (2006–2010): 26, 18, 14, 7, 30
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Correction factor: Based on the Queensland and Northern Territory population	Constant	1.05
Domestically acquired multiplier: Assumed to be 100% domestically acquired	PERT	Minimum, modal, maximum values: 1, 1, 1
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> multiplier adapted from Hall et al (3)	Log Normal	Mean, standard deviation: 7.44, 2.38
Total illness: Reported cases (Queensland notifications) x population adjustment x underreporting(O-S)(S-C) x Proportion travel-related	Outcome	5%, median, 95% values: 40, 150, 300
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 2, 7, 14
Foodborne multiplier: Assumed to be 100% foodborne	PERT	Minimum, modal, maximum values: 1, 1, 1
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 40, 150, 300
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 9% values: 2, 7, 14

Clostridium perfringens

Technical Appendix 4 Table 6. Primary Data: Outbreak; Alternate Data: Water Quality Study

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: The number of <i>C. perfringens</i> outbreak-associated illnesses reported to OzFoodNet 2006–2008.	Empirical	By year (2006–2008): 183, 44, 383
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2008): 20697880, 21015936, 21384427
Domestically acquired multiplier: Assumed to be 100% domestically acquired due to the short incubation period	PERT	Minimum, modal, maximum values: 1, 1, 1
Underreporting: Outbreak multiplier used to adjust from outbreak to surveillance (O-S) Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> multiplier adapted from Hall et al. (3)	PERT Log Normal	Minimum, modal, maximum values: 5, 14, 20 Mean, standard deviation: 7.44, 2.38

Model Input, Source and Comments	Distribution	Data for Model Input
Total illness: Outbreak cases x underreporting(O-S)(S-C) x proportion travel-related	Outcome	5%, median, 95% values: 2600, 16500, 53400
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 35, 785, 2465
Foodborne multiplier: Expert elicitation study 2009	PERT	Minimum, modal, maximum values: 0.86, 0.98, 1
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 2550, 16100, 50600
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 95% values: 130, 765, 2350

***Cryptosporidium* spp.**

Technical Appendix 4 Table 7. Primary Data: National Notifiable Disease Surveillance System (NNDSS); Alternate Data: Water Quality Study

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: NNDSS data. Available from: http://www9.health.gov.au/cda/source/rpt_4.cfm (cited 2013 Nov 12)	Empirical	By year (2006–2010): 3201, 2809, 2004, 4624, 1479
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier: NNDSS travel data	PERT	Minimum, modal, maximum values: 0.92, 0.97, 0.99
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> multiplier adapted from Hall et al. (3)	Log Normal	Mean, standard deviation: 7.44, 2.38
Total illness: Reported cases (NNDSS) x travel adjustment x underreporting (S-C)	Outcome	5%, median, 95% values: 8150, 17900, 39800
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 365, 850, 1860
Foodborne multiplier: Based on 2005 expert elicitation	Alternate PERT	5%, median, 95% values: 0.01, 0.1, 0.27
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 150, 1700, 6100
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 9% values: 57, 80, 320

Giardia lamblia

Technical Appendix 4 Table 8. Primary Data: Victoria Notifications; Alternate Data: Water Quality Study

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: Victorian State notifications from: O'Grady and Tallis (4); Brown et al. (5–8). Giardiasis became a non-notifiable disease in Victoria in 2010	Empirical	By year (1996–2000): 1085, 1060, 999, 921, 866 By year (2006–2009): 1192, 1382, 1434, 1433
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (1996–2000): 18310714, 18517564, 18711271, 18925855, 19153380 By year (2006–2009): 20697880, 21015936, 21384427, 21778845
Correction factor: Based on the Victoria population	Constant	4.03
Domestically acquired multiplier: Victorian notification data (9)	PERT	Minimum, modal, maximum values: 0.84, 0.85, 0.89
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> multiplier adapted from Hall et al (3)	Log Normal	Mean, standard deviation: 7.44, 2.38
Total illness: Reported cases (Victoria notifications) x population adjustment x underreporting (O-S)(S-C) x proportion travel-related	Outcome	5%, median, 95% values: 19800, 32800, 56400
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 920, 1560, 2665
Foodborne multiplier: Based on 2005 expert elicitation	PERT	Minimum, modal, maximum values: 0.01, 0.06, 0.5
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 800, 3700, 10600 (circa 2010) 5%, median, 95% values: 565, 2600, 7400 (circa 2000)
Rate of foodborne illness per million: Circa 2010 and circa 2000	Outcome	5%, median, 9% values: 35, 175, 490 (circa 2010) 5%, median, 9% values: 30, 140, 405 (circa 2000)

Hepatitis A

Technical Appendix 4 Table 9. Primary Data: National Notifiable Disease Surveillance System (NNDSS); Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: NNDSS data. Available from: http://www9.health.gov.au/cda/source/rpt_4.cfm (cited 2013 Nov 12)	Empirical	By year (1996–2000): 2058, 3032, 2466, 1551, 809 By year (2006–2010): 281, 166, 277, 564, 267
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (1996–2000): 18310714, 18517564, 18711271, 18925855, 19153380 By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier: NNDSS travel data	PERT	Minimum, modal, maximum values: 0.42, 0.58, 0.77
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C).	Alternate Pert	2.5%, median, 97.5% values: 1, 2, 3

Model Input, Source and Comments	Distribution	Data for Model Input
Total illness: Reported cases (NNDSS) x travel adjustment x underreporting (S-C)	Outcome	5%, median, 95% values: 150, 300, 800
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 7, 15, 35
Foodborne multiplier: Expert elicitation study 2009	Alternate PERT	5%, median, 95% values: 0.05, 0.12, 0.24
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 10, 40, 100 (circa 2010) 5%, median, 95% values: 65, 245, 725 (circa 2000)
Rate of foodborne illness per million: Circa 2010 and circa 2000	Outcome	5%, median, 9% values: 1, 2, 5 (circa 2010) 5%, median, 9% values: 3, 13, 40 (circa 2000)

*NA, not applicable.

Listeria monocytogenes

Technical Appendix 4 Table 10. Primary Data: National Notifiable Disease Surveillance System (NNDSS); Alternate Data: Outbreak

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: NNDSS data. Available from: http://www9.health.gov.au/cda/source/rpt_4.cfm (cited 2013 Nov 12)	Empirical	By year (1996–2000): 66, 74, 53, 63, 67 By year (2006–2010): 61, 50, 68, 92, 71
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (1996–2000): 18310714, 18517564, 18711271, 18925855, 19153380 By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier: Assumed to be 100% because most of the travelers are not at high risk	PERT	Minimum, modal, maximum values: 1, 1, 1
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C).	Alternate Pert	2.5%, median, 97.5% values: 1, 2, 3
Total illness: Reported cases (NNDSS) x travel adjustment x underreporting (S-C)	Outcome	5%, median, 95% values: 50, 150, 200
Rate of total illness per million: circa 2010	Outcome	5%, median, 95% values: 3, 7, 75
Foodborne multiplier: Expert elicitation study 2009	Alternate PERT	5%, median, 95% values: 0.9, 0.98, 1
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 50, 150, 200 (circa 2010) 5%, median, 95% values: 70, 125, 185 (circa 2000)
Rate of foodborne illness per million: Circa 2010 and circa 2000	Outcome	5%, median, 9% values: 3, 7, 75 (circa 2010) 5%, median, 9% values: 4, 7, 10 (circa 2000)

Norovirus

Technical Appendix 4 Table 11. Primary Data: Water Quality Study; Alternate Data: Outbreak*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness:		
Gastroenteritis multiplier—based on the 2008 National Gastroenteritis Survey	Alternate PERT	2.5%, median, 97.5% values: 0.64, 0.74, 0.84
Pathogen fraction multiplier—based on age adjusted water quality study of an estimated 69 positive isolates per 703 specimens, (Sinclair et al. (10))	Alternate PERT	2.5%, median, 97.5% values: 0.0772, 0.0982, 0.1226
Population adjustment:	Empirical	By year (2006–2010):
Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)		20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier:		NA
All illnesses in the Water Quality Study were domestically acquired		
Time trend multiplier:		NA
No time trend		
Underreporting:		NA
Water Quality Study is community surveillance		
Total illness:	Outcome	5%, median, 95% values: 1220000, 1550000, 1940000
Population at risk x gastroenteritis multiplier x pathogen fraction multiplier x time trend multiplier		
Rate of total illness per million:	Outcome	5%, median, 95% values: 57100, 72500, 90550
Circa 2010		
Foodborne multiplier:	Alternate PERT	5%, median, 95% values: 0.05, 0.18, 0.35
Expert elicitation study 2009		
Total foodborne illness:	Outcome	5%, median, 95% values: 78100, 276000, 563000
Total illness x foodborne multiplier		
Rate of foodborne illness per million:	Outcome	5%, median, 95% values: 3620, 12920, 26300
Circa 2010		

*NA, not applicable.

Other pathogenic *Escherichia coli*

Technical Appendix 4 Table 11. Primary Data: Water Quality Study; Alternate Data: IID2*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness:		
Gastroenteritis multiplier—based on the 2008 National Gastroenteritis Survey	Alternate PERT	2.5%, median, 97.5% values: 0.64, 0.74, 0.84
Pathogen fraction multiplier—based on age adjusted water quality study of an estimated 50 positive isolates per 713 specimens, (Hellard et al [1])	Alternate PERT	2.5%, median, 97.5% values: 0.0525, 0.074, 0.0914
Population adjustment:	Empirical	By year (2006–2010):
Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)		20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier:		NA
All illnesses in the Water Quality Study were domestically acquired		

Model Input, Source and Comments	Distribution	Data for Model Input
Time trend multiplier: No time trend		NA
Underreporting: Water Quality Study is community surveillance		NA
Total illness: Population at risk x gastroenteritis multiplier x pathogen fraction multiplier x time trend multiplier	Outcome	5%, median, 95% values: 833000, 1100000, 1450000
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 39150, 51350, 67550
Foodborne multiplier: Expert elicitation study 2009	Alternate PERT	5%, median, 95% values: 0.08, 0.23, 0.55
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 85800, 255000, 632000
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 95% values: 4100, 11600, 29700

*Longitudinal study of infectious intestinal disease in the UK. NA, not applicable.

Rotavirus

Technical Appendix 4 Table 11. Primary Data: Water Quality Study; Alternate Data: IID2*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: Gastroenteritis multiplier—based on the 2008 National Gastroenteritis Survey	Alternate PERT	2.5%, median, 97.5% values: 0.64, 0.74, 0.84
Pathogen fraction multiplier—based on age adjusted water quality study of an estimated 50 positive isolates per 713 specimens, (Hellard et al. [1])	Alternate PERT	2.5%, median, 97.5% values: 0.0031, 0.0084, 0.0182
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier: All illnesses in the Water Quality Study were domestically acquired		NA
Time trend multiplier: Based on Dey et al. (11)	Alternate PERT	2.5%, median, 97.5% values: 0.318, 0.338, 0.359
Underreporting: Water Quality Study is community surveillance		NA
Total illness: Population at risk x gastroenteritis multiplier x pathogen fraction multiplier x time trend multiplier	Outcome	5%, median, 95% values: 18500, 44800, 90800
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 875, 2100, 4260
Foodborne multiplier: Expert elicitation study 2009	Alternate PERT	5%, median, 95% values: 0.01, 0.02, 0.03
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 300, 850, 2000

Model Input, Source and Comments	Distribution	Data for Model Input
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 95% values: 15, 40, 95

*Longitudinal study of infectious intestinal disease in the UK. NA, not applicable.

***Salmonella* spp., nontyphoidal (refers to nontyphoidal *Salmonella enterica* serotypes)**

Technical Appendix 4 Table 14. Primary Data: National Notifiable Disease Surveillance System (NNDSS); Alternate Data: Water Quality Study

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: NNDSS data. Available from: http://www9.health.gov.au/cda/source/rpt_4.cfm (cited 2013 Nov 12)	Empirical	By year (1996–2000): 5744, 6955, 7513, 7008, 6187 By year (2006–2010): 8241, 9502, 8316, 9524, 11928
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (1996–2000): 18310714, 18517564, 18711271, 18925855, 19153380 By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier: NNDSS travel data	PERT	Minimum, modal, maximum values: 0.7, 0.85, 0.95
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C)	Log Normal	Mean, standard deviation: 7.44, 2.38
Total illness: Reported cases (NNDSS) x travel adjustment x underreporting(S-C)	Outcome	5%, median, 95% values: 31900, 56200, 101000
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 1515, 2650, 4650
Foodborne multiplier: Expert elicitation study 2009	Alternate PERT	5%, median, 95% values: 0.53, 0.72, 0.86
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 21200, 39600, 73400 (circa 2010) 5%, median, 95% values: 15000, 28000, 50000 (circa 2000)
Rate of foodborne illness per million: Circa 2010 and circa 2000	Outcome	5%, median, 9% values: 1000, 1850, 3350 (circa 2010) 5%, median, 9% values: 800, 1500, 2700 (circa 2000)

***Salmonella enterica* serotype Typhi**

Technical Appendix 4 Table 15. Primary Data: National Notifiable Disease Surveillance System (NNDSS); Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: NNDSS data. Available from: http://www9.health.gov.au/cda/source/rpt_4.cfm (cited 2013 Nov 12)	Empirical	By year (1996–2000): 72, 72, 57, 63, 58 By year (2006–2010): 77, 90, 105, 115, 95
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (1996–2000): 18310714, 18517564, 18711271, 18925855, 19153380 By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier: NNDSS travel data	PERT	Minimum, modal, maximum values: 0.02, 0.11, 0.25
Underreporting:	Alternate	2.5%, median, 97.5% values: 1, 2, 3

Model Input, Source and Comments	Distribution	Data for Model Input
Multiplier used to adjust for underreporting from surveillance to community (S-C)	PERT	
Total illness: Reported cases (NNDSS) x travel adjustment x underreporting (S-C)	Outcome	5%, median, 95% values: 8, 20, 45
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 0, 1, 2
Foodborne multiplier: Based on 2005 expert elicitation	PERT	Minimum, modal, maximum values: 0.02, 0.75, 0.97
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 5, 15, 30 (circa 2010) 5%, median, 95% values: 3, 9, 21 (circa 2000)
Rate of foodborne illness per million: Circa 2010 and circa 2000	Outcome	5%, median, 9% values: 0, 0.6, 1 (circa 2010) 5%, median, 9% values: 0, 0.5, 1 (circa 2000)

*NA, not applicable.

Sapovirus

Technical Appendix 4 Table 16. Primary Data: Water Quality Study; Alternate Data: IID2*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: Gastroenteritis multiplier—based on the 2008 National Gastroenteritis Survey	Alternate PERT	2.5%, median, 97.5% values: 0.64, 0.74, 0.84
Pathogen fraction multiplier—based on age adjusted water quality study findings for norovirus of an estimated 69 positive isolates per 703 specimens (Sinclair et al. [10])	Alternate PERT	2.5%, median, 97.5% values: 0.0772, 0.0982, 0.1226
Pathogen comparison multiplier – Kirkwood multiplier (2) comparing norovirus to sapovirus	Constant	0.5
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier: All illnesses in the Water Quality Study were domestically acquired		NA
Time trend multiplier: No time trend		NA
Underreporting: Water Quality Study is community surveillance		NA
Total illness: Population at risk x gastroenteritis multiplier x pathogen fraction multiplier x time trend multiplier	Outcome	5%, median, 95% values: 63400, 81600, 102000
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 3000, 3800, 4800
Foodborne multiplier: Assumed to be the same as norovirus	PERT	Minimum, modal, maximum values: 0.05, 0.18, 0.35
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 7450, 15000, 24300
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 95% values: 350, 700, 1150

*Longitudinal study of infectious intestinal disease in the UK. NA, not applicable.

Scombrototoxicosis

Technical Appendix 4 Table 17. Primary Data: Outbreak; Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: The number of scombrototoxicosis outbreak-associated illnesses reported to OzFoodNet 2006–2008.	Empirical	By year (2006–2008): 12, 17, 0
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2008): 20697880, 21015936, 21384427
Domestically acquired multiplier: Assumed to be 100% domestically acquired due to the short incubation period	PERT	Minimum, modal, maximum values: 1, 1, 1
Underreporting: Outbreak multiplier used to adjust from outbreak to surveillance (O-S) Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> multiplier adapted from Hall et al (3)	PERT Log Normal	Minimum, modal, maximum values: 5, 14, 20 Mean, standard deviation: 7.44, 2.38
Total Illness: Outbreak cases x underreporting (O-S)(S-C) x proportion travel-related	Outcome	5%, median, 95% values: 0, 1050, 2450
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 0, 50, 115
Foodborne multiplier: Assumed to be 100% foodborne	PERT	Minimum, modal, maximum values: 1, 1, 1
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 0, 1050, 2450
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 95% values: 0, 50, 115

*NA, not applicable.

Shigella spp.

Technical Appendix 4 Table 17. Primary Data: National Notifiable Disease Surveillance System (NNDSS); Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: NNDSS data. Available from: http://www9.health.gov.au/cda/source/rpt_4.cfm (cited 2013 Nov 12)	Empirical	By year (1996–2000): 660, 802, 580, 534, 488 By year (2006–2010): 545, 597, 828, 618, 550
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (1996–2000): 18310714, 18517564, 18711271, 18925855, 19153380 By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Domestically acquired multiplier: NNDSS travel data	PERT	Minimum, modal, maximum values: 0.45, 0.7, 0.84
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> spp. multiplier adapted from Hall et al. (3)	Log Normal	Mean, standard deviation: 7.44, 2.38
Total Illness: Reported cases (NNDSS) x travel adjustment x underreporting (S-C)	Outcome	5%, median, 95% values: 1650, 3000, 5400
Rate of total illness per million:	Outcome	5%, median, 95% values: 75, 140, 260

Model Input, Source and Comments	Distribution	Data for Model Input
Circa 2010		
Foodborne multiplier: Expert elicitation study 2009	Alternate PERT	5%, median, 95% values: 0.05, 0.12, 0.23
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 150, 350, 850 (circa 2010) 5%, median, 95% values: 175, 515, 1300 (circa 2000)
Rate of foodborne illness per million: Circa 2010 and circa 2000	Outcome	5%, median, 9% values: 6, 16, 40 (circa 2010) 5%, median, 9% values: 9, 28, 70 (circa 2000)

*NA, not applicable.

Staphylococcus aureus

Technical Appendix 4 Table 19. Primary Data: Outbreak; Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: The number of <i>S. aureus</i> outbreak-associated illnesses reported to OzFoodNet 2006–2008	Empirical	By year (2006–2008): 3, 14, 50
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2008): 20697880, 21015936, 21384427
Domestically acquired multiplier: Assumed to be 100% domestically acquired due to the short incubation period	PERT	Minimum, modal, maximum values: 1, 1, 1
Underreporting: Outbreak multiplier used to adjust from outbreak to surveillance (O-S) Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> multiplier adapted from Hall et al. (3)	PERT Log Normal	Minimum, modal, maximum values: 5, 14, 20 Mean, standard deviation: 7.44, 2.38
Total Illness: Outbreak cases x underreporting (O-S)(S-C) x proportion travel-related	Outcome	5%, median, 95% values: 200, 1300, 7050
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 9, 60, 350
Foodborne multiplier: Based on 2005 expert elicitation	PERT	Minimum, modal, maximum values: 0.95, 1, 1
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 200, 1300, 7000
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 95% values: 9, 60, 350

*NA, not applicable.

Shiga toxin–producing *Escherichia coli*

Technical Appendix 4 Table 20. Primary Data: South Australian Surveillance; Alternate Data: National Notifiable Disease Surveillance System

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: South Australian State STEC surveillance from the study by Vally et al. (12)	Empirical	By year (2006–2010): 35, 40, 39, 62, 32
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Correction factor: Based on the South Australian population	Constant	13.4
Domestically acquired multiplier: NNDSS travel data	PERT	Minimum, modal, maximum values: 0.93, 0.99, 1
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C). STEC multiplier adapted from Hall et al (3)	Log Normal	Mean, standard deviation: 8.83, 3.7
Total illness: Reported cases(SA surveillance) x correction factor x travel adjustment x underreporting (S-C)	Outcome	5%, median, 95% values: 2050, 4300, 9500
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 100, 200, 450
Foodborne multiplier: Expert elicitation study 2009	Alternate PERT	5%, median, 95% values: 0.32, 0.56, 0.83
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 950, 2350, 5850
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 9% values: 45, 110, 260

Toxoplasma gondii

Technical Appendix 4 Table 21. Primary Data: State and Territory Notifications; Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: US seroprevalence data (13) extrapolated to the Australian population for 2010 by age group	Empirical	0-4: 5709 5-9: 5749 10-19: 10744 20-29: 11728 30-39: 10809 40-49: 10377 50-59: 8903 60-69: 6521 70-79: 3713 80+: 2342 Total: 76095
Population adjustment: Australian resident population 2010 by age group June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument	Empirical	0-4: 1441679 5-9: 1352211 10-19: 2852050

Model Input, Source and Comments (cited 2012 Aug 16)	Distribution	Data for Model Input
		20-29: 3240347 30-39: 3108224 40-49: 3105877 50-59: 2773511 60-69: 2114158 70-79: 1253114 80+: 824146
Domestically acquired multiplier: Assumed to be 100% domestically acquired	PERT	Minimum, modal, maximum values: 1, 1, 1
Proportion symptomatic: Scallan et al. (14) and Abelson et al. (15)	PERT	Minimum, modal, maximum values: 0.11, 0.15, 0.21
Total illness: Estimated yearly cases x travel adjustment x proportion symptomatic	Outcome	5%, median, 95% values: 8350, 11400, 16000
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 380, 515, 760
Foodborne multiplier: Based on 2005 expert elicitation	PERT	Minimum, modal, maximum values: 0.04, 0.31, 0.74
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 1400, 3750, 7150
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 9% values: 65, 170, 325

*NA, not applicable.

Vibrio parahaemolyticus

Technical Appendix 4 Table 22. Primary Data: Western Australia Notifications; Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: Western Australia Notifications— http://www.public.health.wa.gov.au/cproot/4195/2/12172_DiseaseWatch.pdf	Empirical	By year (2006–2010): 3, 9, 7, 9, 10
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Correction factor: Based on the Western Australia population	Constant	9.61
Domestically acquired multiplier: OzFoodNet WA Annual Reports 2006–2010	PERT	Minimum, modal, maximum values: 0, 0.18, 0.33
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> multiplier adapted from Hall et al. (3)	Log Normal	Mean, standard deviation: 7.44, 2.38
Total Illness: Reported cases (Western Australia notifications) x population adjustment x underreporting (O-S)(S-C) x proportion travel-related	Outcome	5%, median, 95% values: 15, 60, 170
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 1, 3, 8

Model Input, Source and Comments	Distribution	Data for Model Input
Foodborne multiplier: Based on 2005 expert elicitation	PERT	Minimum, modal, maximum values: 0.05, 0.75, 0.96
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 10, 40, 120
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 9% values: 0, 2, 6

*NA, not applicable.

Yersinia enterocolitica

Technical Appendix 4 Table 23. Primary Data: State and Territory Notifications; Alternate Data: NA*

Model Input, Source and Comments	Distribution	Data for Model Input
Reported illness: State notifications from Queensland, South Australia, Western Australia, and Northern Territory extrapolated from State data to the Australian population to determine the expected number of notifications if all States were reporting	Empirical	By year (2006–2010): 214, 249, 326, 242, 239
Population adjustment: Australian resident population 2006–2010 June quarter http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202011?OpenDocument (cited 2012 Aug 16)	Empirical	By year (2006–2010): 20697880, 21015936, 21384427, 21778845, 22065317
Correction factor: Based on the Western Australia population	Constant	9.61
Domestically acquired multiplier: OzFoodNet Western Australia Annual Reports 2006–2010	PERT	Minimum, modal, maximum values: 0.8, 0.9, 1
Underreporting: Multiplier used to adjust for underreporting from surveillance to community (S-C). Nontyphoidal <i>Salmonella</i> multiplier adapted from Hall et al (3)	Log Normal	Mean, standard deviation: 7.44, 2.38
Total Illness: Reported cases (extrapolated State notifications) x population adjustment x underreporting (O-S)(S-C) x proportion travel-related	Outcome	5%, median, 95% values: 1900, 1500, 2500
Rate of total illness per million: Circa 2010	Outcome	5%, median, 95% values: 140, 70, 115
Foodborne multiplier: Based on 2005 expert elicitation	PERT	Minimum, modal, maximum values: 0.28, 0.84, 0.94
Total foodborne illness: Total illness x foodborne multiplier	Outcome	5%, median, 95% values: 650, 1150, 1950
Rate of foodborne illness per million: Circa 2010	Outcome	5%, median, 9% values: 30, 50, 90

*NA, not applicable.

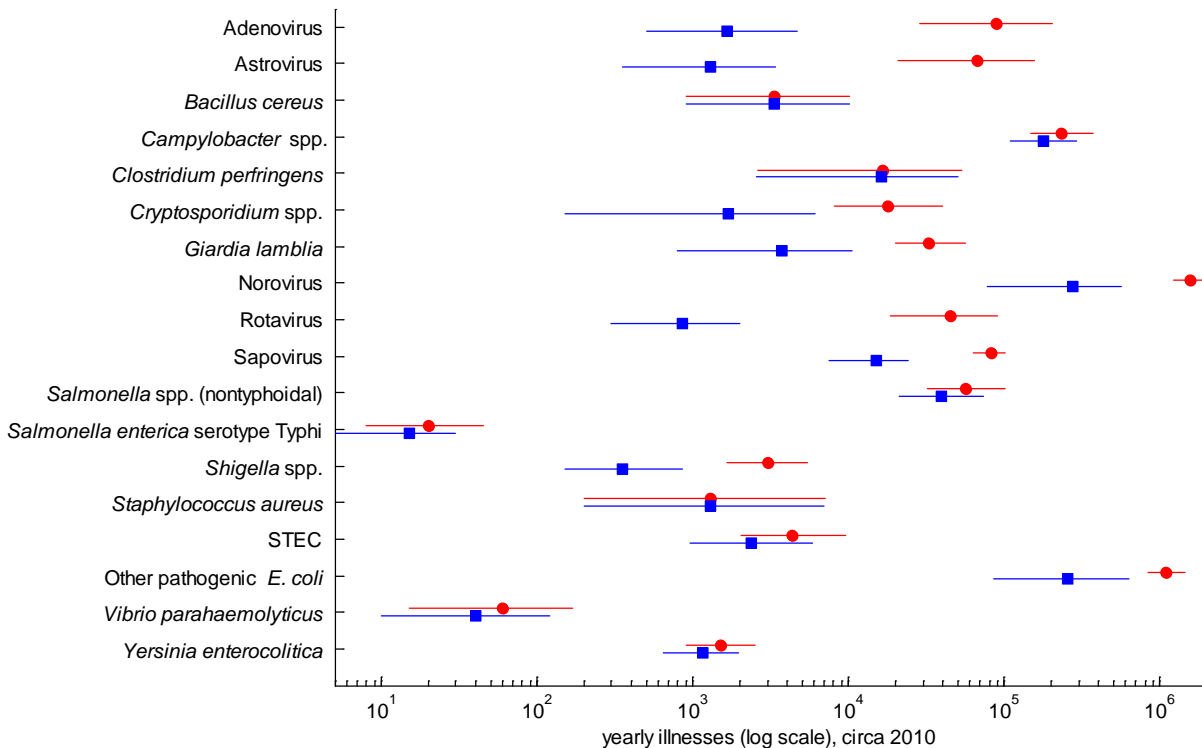
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Foodborne Illness, Australia, Circa 2000 and Circa 2010

Technical Appendix 5



Technical Appendix 5 Figure. Median number of all domestically acquired illnesses (red dots) and domestically acquired foodborne illnesses (blue squares), by pathogen, Australia, circa 2010. Bars indicate 90% credible intervals. *E. coli*, *Escherichia coli*; STEC, Shiga toxin-producing *E. coli*. *Salmonella* spp. (nontyphoidal) refers to nontyphoidal *Salmonella enterica* serotypes.